# Bone Morphologies and Histories: Life Course Approaches in Bioarchaeology

Sabrina C. Agarwal*

*Department of Anthropology, University of California Berkeley, Berkeley, CA 94720-3710*

## ABSTRACT

The duality of the skeleton as both a biological and cultural entity has formed the theoretical basis of bioarchaeology. In recent years bioarchaeological studies have stretched the early biocultural concept with the adoption of life course approaches in their study design and analyses, making a significant contribution to how we think about the role of postnatal plasticity. Life course theory is a conceptual framework used in several scientific fields of biology and the social sciences. Studies that emphasize life course approaches in the examination of bone morphology in the past are united in their interrogation of human life as a result of interrelated and cumulative events over not only the timeframe of individuals, but also over generations at the community level. This article provides an overview of the theoretical constructs that utilize the life course concept, and a discussion of the different ways these theories have been applied to thinking about trajectories of bone morphology in the past, specifically highlighting key recent studies that have used life course approaches to understand the influence of growth, stress, diet, activity, and aging on the skeleton. The goal of this article is to demonstrate the scope of contemporary bioarchaeological studies that illuminate the importance of environmental and behavioral influence on bone morphology. Understanding how trajectories of bone growth and morphology can be altered and shaped over the life course is critical not only for bioarchaeologists, but also researchers studying bone morphology in living nonhuman primates and fossil primate skeletons. Am J Phys Anthropol 159:S130–S149, 2016.    © 2016 Wiley Periodicals, Inc.

## INTRODUCTION

The study of human biology and behavior in biological anthropology has its foundation in the study of variation in morphology, particularly of the skeleton. The history of physical anthropology began with a focus on biological (particularly racial) differences in skeletal morphology in the 18th and 19th centuries, and while the evolutionary approaches of the "New Physical Anthropology" shifted focus to the study of living primates and fossil non-human and human primates, the study of variation in skeletal morphologies continued to be fundamental to the field (Little and Kennedy, 2010). With advances in molecular and cellular biology, contemporary biological anthropology is now a methodologically wider field, and evolutionary approaches to human variation now share interdisciplinary overlap with subdisciplines such as human biology, genetic anthropology, medical anthropology, and evolutionary psychology. Simultaneously, our understanding of the evolution of mammalian and human skeletal morphology has been radically expanded with insights from evolutionary developmental biology (Lovejoy et al., 2003). In the past decade developmental biology has increasingly grounded questions of phylogeny and functional adaptation in human and nonhuman primate evolution. This research has led to the use of more complex and multifactorial explanations of the role of developmental-genetic interactions on variation in skeletal morphology, such as the use of experimental models in the understanding of variation in cranio-facial morphology (Martínez-Abadías et al., 2012), and in variation in the ontogeny of the post-cranial skeleton (Hallgrimsson et al., 2002b; Ryan and Krovitz, 2006; Gosman and Ketcham, 2009).

Alongside these fundamental breakthroughs in developmental biology, studies of variation by human biologists, paleopathologists, and particularly bioarchaeologists, have concentrated on the examination of variation in morphology related to influences such as activity, disease, and nutrition. Bioarchaeological studies focus largely on the morphology of anatomically modern humans and are interested in variation within human populations and the level of the individual, rather than wider evolutionary questions of phylogenetic variation. Bioarchaeology is rooted in the early biocultural approach with an emphasis on archaeological contextualization (Armelagos, 2003; Buikstra and Beck, 2009) and concerns the interaction of bone biology and behavior along with environmental influences on health and lifestyle (Larsen, 2002). Contemporary work has also emphasized the need to tie research to relevance in modern populations and incorporate critical social theory in interpretations (Agarwal and Glencross, 2011; Martin et al., 2014). In recent years, bioarchaeological studies have made relevant contributions to our wider understanding of bone morphology with the adoption of life course perspectives in the analysis of morphology at the individual and population-levels. Life course approaches are a widely used and familiar theoretical orientation in sociological and epidemiological studies

*Correspondence to: Sabrina Agarwal, Department of Anthropology, University of California Berkeley, 232 Kroeber Hall, Berkeley, CA 94720-3710. E-mail: agarwal@berkeley.edu

(Ben-Schlomo and Kuh, 2002; Elder et al., 2003; Mayer, 2009). While only a small number of bioarchaeological studies have explicitly cited life course approaches, studies of bone morphology in the past are united in their interrogation of human life as a result of interrelated and cumulative events over not only the timeframe of individuals, but also at the community level over generations. These studies are, however, not oblivious to the role of evolutionary developmental biology or the complex genetic basis of bone morphology. Many contemporary bioarchaeological studies are ultimately interested in understanding the interaction of environmental and genetic influences on phenotypic variation. These studies stretch the concept of the early biocultural approach in the field, and make a significant contribution to how we think about the role of postnatal plasticity.

In this article I provide an overview of the theoretical constructs in biology and the social sciences that utilize the life course concept, and how these relate to evolutionary developmental biology and plasticity, as well as lateral concepts such as developmental systems theory and embodiment theory. I then discuss how these theories have been considered in studies on bone morphology[1], working through the key studies both historically and in contemporary bioarchaeology that utilize life course approaches in its varied forms. Spanning growth studies, paleopathology and archaeological approaches to embodiment, these studies demonstrate the importance of biosocial approaches to plasticity and development. Finally, I will take a focused look at how life course approaches can be used to more holistically understand one key aspect of bone morphology, specifically bone growth, maintenance and loss. While some anthropological perspectives have dismissed studies that emphasize postnatal plasticity in bone morphology (Lovejoy et al., 2003), the goal of this article is to cohesively demonstrate the scope of bioarchaeological studies that illuminate the importance of environmental and behavioral influence on bone morphology. With their emphasis on the cumulative role of experiences across the life course and potentially across generations these studies have much to offer to our understanding of bone morphology in the past and the present.

## LIFE COURSE THEORIES AND DEVELOPMENTAL APPROACHES TO PLASTICITY

Life course theory is a conceptual framework that has been generated and used in several scientific fields including biology and the social sciences. The heart of the paradigm is the study of individual lives and their connection to their historical and socioeconomic contexts. I include here, under the umbrella of life course, all approaches that view the individual at any point in time as the sum of previous life experiences. These include both social and biological experiences, and the individual plastic response to exposures can be viewed as adaptive or not, and can be viewed as intergenerational or only affecting the phenotypic expression in the individual.

In sociology, researchers use life course theory to better understand the construction of social pathways, and how institutions and historical events shape and link individual and group experiences (Clausen, 1985; Elder, 1995). While individual development is key, individual agency is also framed as exerting influence on, and being influenced by, the given social structure (Bengston and Allen, 1993). Interdependency is the key component, whereby development at the micro-level of the individual and families are placed within interconnected contexts that extend to the biographies of generations over larger periods of time and space (Bengston and Allen, 1993).

Health researchers also became interested in how this framework could relate to the development of health and disease. Contemporary models of life course health development are based on current findings in epigenetics, systems biology and longitudinal studies. In epidemiology, emphasis is on the role of physical and social exposures during gestation, childhood, adolescence, and young adulthood, and the physical manifestations of disease in later adult life (Halfon et al., 2014). Early life factors in particular have been shown to increase the risk of a broad spectrum of diseases (Ben-Schlomo and Kuh, 2002). This phenomenon has also been termed the Developmental Origins of Health and Disease (DOHaD) paradigm, which posits that early life environmental events, in association with genetic predisposition, play a central role in influencing responses to the environment and thus later disease risk. Particularly influential was the pioneering work on early fetal and childhood environment to later adult health. For example, the "Barker hypothesis" suggested that poor nutrition during pregnancy could alter "fetal programming" that could change the course of the developing body's structure, function or metabolism (Barker et al., 1989; Kuh and Davey Smith, 2004). This research by Barker and colleagues first linked birth weight to the risk of cardiovascular disease in later life (Barker and Osmond, 1986; Barker et al., 1989; Benyshek, 2013). Gluckman et al. later elaborated that conditions such poor nutrition during pregnancy results in a "predictive adaptive response" (PAR) in the fetus whereby developmental pathways are developed that are perceived to be better suited to the postnatal environment (Gluckman and Hanson, 2004; Low et al., 2012). This observation has also been called the "thrifty phenotype" model of human life history and argues that these predictive physiological responses, and often resulting diseases, reflect a mismatch between the fetal development and the actual postnatal environmental landscape (Hales and Barker, 2001). These developing physiological pathways can lead to predictive responses such as increased fat storage or reduced insulin production, but when nutrition is plentiful, postnatally the mismatch can lead to diabetes (Halfon et al., 2014).

Key to life course approaches are the concepts of trajectories and plasticity. First, development is conceived as following an arc of development or trajectory that can change direction along the phases of the life course. In portraying development as a trajectory it is possible to imagine the range of different influences that alter development in different outcomes (Low et al., 2012; Halfon et al., 2014). The idea is that development will alter trajectory as needed as an adaptive process. Further, operating over long periods of time, evolutionary forces have selected trajectories and strategies that have plasticity to adapt to a range of environments (Halfon et al., 2014). Plasticity is the second key concept in life course approaches. Plasticity is related to both the concepts of growth and development that together form the

---

[1] I use the term bone *morphology* here broadly, referring to the form and structure of individual bony elements/tissues or whole skeletons including outward appearance such as size and shape, but also internal structure, microstructure and chemical structure.

foundation to understanding patterns of phenotypic variability (Agarwal and Beauchesne, 2011). Growth is the enlargement and differentiation of tissues advancing with chronological age, while development comprises the pathways of biological milestones along the life cycle. The rates of growth and timing of developmental changes can differ among individuals and there has been debate over growth and development trajectories and their evolutionary and adaptive significance (Lasker, 1969; Roberts, 1995; Schell, 1995; Bogin, 1999; Ellison, 2005; Worthman and Kuzara, 2005).

Plasticity relates to the ability of an organism to change its phenotype in response to changes in the environment, and it has been conceptually linked to adaptation (Lasker, 1969; Roberts, 1995). Before the 1950s and 1960s plasticity was simply an understanding that human morphology appeared to be malleable during growth and development (Bogin, 1995). Lasker (1976) redefined the plasticity concept with adaptation, and viewed plasticity as operating within three modes of adaptation. The first mode was natural selection itself, where the selection of genotypes directly influences the genetic spectrum of the population (Roberts, 1995). The second form of adaptive plasticity is acclimatization, the physiological and behavioral response that adapts an individual to the immediate environment that is nonpermanent (Roberts, 1995). The third mode that is relevant to our discussion of life course approaches is developmental or ontogenetic adaptation (Roberts, 1995). Here the plasticity operates through growth and development, and it is generally regarded that the changes are not reversible and also not heritable (Schell, 1995). Contemporary studies of variation in human phenotypes through the lens of plasticity are now primarily linked to adaptation and the consideration of biological trade-offs (Worthman and Kuzara, 2005). In this model, the biological response to early fetal or childhood life-stress is seen as a life history trade-off where early stresses such as poor nutrition cause energetic reserves to be reallocated to the development of essential tissues or functions. While early mortality may be thwarted, the result is a trade-off with reduced future investment in growth, higher mortality or impaired fertility (Kuzawa, 2005; Worthman and Kuzara, 2005).

Understanding plasticity in the developmental context beyond a strictly adaptationist model has also been put forward by a number of researchers as developmental systems theory or approach (DST/DSA), and developmental dynamics (Oyama, 2000a; Lewontin, 2001). These life course approaches are also focused on the plastic developmental responses that take place in embryogenesis, during fetal growth, early postnatal growth, and later adolescence. Unlike epidemiological approaches that are primarily focused on plasticity during fetal development or early infancy, DST/DSA approaches extend plasticity to include infancy, childhood, and adolescence (Fausto-Sterling, 2005). Developmental information is believed to reside neither in the genes nor the environment, but rather in the interaction of the two (Roberts, 1995). Plasticity is the essential feature of the development system that is defined as the interplay of all influences on development including the "molecular, cellular, organismal, ecological, social, and biogeographical" (Robert et al. 2001:954). As such development is seen to extend well into postnatal growth (Robert et al., 2001; Worthman and Kuzara, 2005). There are a number of examples of this, including neurological growth (Kamm et al., 1990) and immune

functions (Worthman, 1995). The concept of the body as a product of both biological and social developmental contexts is also found in perspectives on embodied life experience. In this approach embodiment refers to how beings literally incorporate the world in which they exist biologically, including social and ecological variables (Krieger, 2001, 2005). The emphasis is on the process of creation or transformation of beings and organisms over time as a product of engagement with the world in which they are situated. This engagement with the world can be both conscious (with agency) or unconscious (Krieger, 2001, 2005), and challenges the contention that organisms are primarily passively built by their genetic code. The construct of embodiment has been advocated in epidemiology, particularly in better understanding population rates of morbidity and mortality, and social inequalities in health (Krieger, 2001, 2005). Anthropologists have also discussed the human body as a developmental system that is contextually dependent, and have emphasized that humans grow and are active in their development through engagement with the social world (Gravlee, 2009; Ingold, 1998; Joyce, 2005; Sofaer, 2006a).

There have been comparisons between the different life course approaches, particularly developmental systems theory (DST) and evolutionary developmental biology (EDB) (Robert et al., 2001; Hallgrimsson et al., 2002a). EDB perspectives are primarily limited to embryology/fetal development and are less concerned with postnatal plasticity (Robert et al., 2001; Hallgrimsson et al., 2002a). The biggest difference between EDB and life course approaches has been the emphasis on genes. EDB has been classically thought of as giving primacy to the gene, with variation in developmental processes studied in the context of conservation and constraint of form (Robert et al., 2001). However, in recent years there has been more common ground linking developmental approaches through epigenetics. Epigenetics can be defined as the study of changes in organisms caused by modification of gene expression rather than alteration of the genetic code itself. There are three primary known systems considered to initiate and sustain epigenetic change: DNA methylation, histone modification and non-coding RNA (ncRNA)-associated gene silencing (Holliday, 2006). Epigenetic studies have provided clues to the mechanisms that explain the findings of studies in fetal programing and DOHaD research (Halfon et al., 2014). These studies have shown how gene expression can be altered by various environmental influences, particularly in neurodevelopment with intergenerational impacts (Halfon et al., 2014). For example, the recent widely known study by Dias and Ressler (2014) suggested that mice trained to fear the smell of acetophenone were able to pass this fear to their offspring. While transgenerational epigenetic effects still remain controversial there have been many studies of epigenetic inheritance found across multiple plant and animal species (Jablonka and Raz, 2009). In approaches like developmental systems theory (DST) inheritance is extended to include ecological and social resources, or other influences on development (Oyama, 2000b). Epigenetic processes are seen as heritable and are constructed and reconstructed during each life cycle. While there is still uncertainty if or how non-genetic influences are inherited, particularly in relation to skeletal morphology, there is increasing evidence that nongenetic forces can

significantly shape postnatal and skeletal morphology (Fausto-Sterling, 2005; Agarwal and Beauchesne, 2011).

## EXAMINING PLASTICITY AND LIFE HISTORIES IN BIOARCHAEOLOGY

There have been many studies in bioarchaeology that have utilized life course approaches to understand bone morphology and development. While studies have classically utilized population level data with a focus on indicators of early life stress, newer studies in bioarchaeology have included a focus on individual life history, the use of multiple indicators, and a greater concern for approaches that focus on influences over the broader life cycle. Included here are both innovative theoretical and methodological approaches to bone morphology. While the studies included here are varied, they all share an emphasis on the role postnatal plasticity on bone morphology, and an overall aim to better elucidate the synergistic role of biological and environmental influences over the life span, and possibly over generations.

### Development and stress markers

Historically, the study of plasticity in anthropology was taken up by Boas (1912) and in even earlier studies that noted generational changes in growth in migrant studies (Baxter, 1875; Bowditch, 1879). Boas' (1912) detailed anthropometric study of body size and shape in the children of new immigrants of European descent was pivotal in demonstrating that environmental changes including changing cultural milieus could produce changes in body size and shape in future generations. Boas's work was later supported by Shapiro's (1939) well cited growth study of Japanese children in Japan and Hawaii that also showed changes in growth, stature, and development that were attributed to environmental triggers. Since then, numerous migrant studies have confirmed the correlation of changing environments to changes in growth and development (Goldstein, 1943; Lasker and Evans, 1961; Kasl and Berkman, 1983; Baker et al., 1986; Bogin, 1995; Bogin and Rios, 2003). Perhaps the greatest attention to skeletal postnatal plasticity has been devoted to plasticity in growth as a biological outcome of environmental and behavioral stress. Differences in human height or stature are highly heritable and genomic studies have shown hundreds of genes associated with the expression of stature (Lai, 2006; Allen et al., 2010). However, genetic potential is mediated by environmental factors. Perturbations in growth are thought of as a compensatory mechanism to disruptions in homeostasis (Martorell, 1989; Bogin, 1999). While the value of the compensatory response is survival, such developmental adjustments often come at a cost, and individuals that do not meet optimal growth or size have increased risk of morbidity and mortality (Bogin, 1995; Blackwell et al., 2001).

In this way, slowing or stunting growth allows developing organisms to reroute nutrition to other more pressing energetic areas, such as brain growth (Leonard and Robertson, 1992; Bogin, 1995). These perturbations to growth are considered nonspecific, as they can have numerous etiologies including insults from malnutrition, infection or psychosocial influences (Saunders and Hoppa, 1993; Blackwell et al., 2001).

The examination of stature derived from long bone length, and other nonspecific indicators of stress, have been used to examine early life stress and their link to morbidity and mortality in skeletal populations. Other nonspecific "stress" indicators include a variety of skeletal and dental markers such as dental enamel defects, fluctuating asymmetry in bilateral bone structures, and changes to vertebral neural canal size. Like stunting or slowing in growth of long bones, these other nonspecific indicators of stress reflect a physiological disruption (Larsen, 2015). Populations that show reduced stature show evidence of greater systemic stress than those without reduced stature (Bogin, 1995; Saunders, 2000; Stinson, 2000); as such bioarchaeologists have typically used stature as an indicator of overall health and stress during growth (Saunders and Hoppa, 1993; Hoppa and FitzGerald, 2005). Patterns of long bone growth in archaeological skeletal samples have been widely used as a proxy for comparing health and stress statuses between and among populations (Humphrey; Saunders, 2000; Kemkes-Grottenthaler, 2005; Mays et al., 2008; Lewis, 2009). Early bioarchaeological studies were of general physiological stress responses, with a focus on stress as an adaptive response (Temple and Goodman, 2014). A number of early classic and recent bioarchaeological investigations have specifically noted early mortality associated with small stature, as well as with stress indicators such as tooth enamel defects (linear enamel hypoplasia) (for example, Buikstra and Cook, 1980; Goodman et al., 1988; Duray, 1996; Humphrey and King, 2000; Steckel, 2005; DeWitte and Wood, 2008) and smaller vertebral neural canal size (Clark et al., 1986; Watts, 2011). While older studies also examined stress markers such growth arrest lines (Harris lines), periosteal new bone formation (periostitis), and porosity/pitting on cranial vaults (porotic hyperostosis and cribra orbitalia), recent studies have focused on examining several of these stress indicators together or in conjunction with dental defects or reduced stature (for example, McHenry, 1968; Klaus and Tam, 2009; Temple, 2010; Turner and Armelagos, 2012; Wheeler, 2012; Watts, 2013; Geber, 2014; Vercellotti et al., 2014; Scott and Hoppa, 2015).

Contemporary studies have made a substantial shift that squarely focuses on developmental plasticity and overt attention to life course approaches. This research also emphasizes the potential for differential severity of stress on adult skeletal morphology and mortality and the potential for recovery. Further, the simultaneous study of multiple skeletal indicators allows the study of stress at longitudinally different points along the life cycle even within the limits of cross-sectional skeletal data. For example, early developmental stress indicators such as linear enamel hypoplasia on the anterior tooth crowns can reflect development between one and six years of age, whereas as cribra orbitalia observed on adult frontal bones is thought to reflect development before four years of age, and long bone lengths reflect growth in utero up to late adolescence and early adulthood (Watts, 2013). Watts (2013) utilized this explicit approach in her examination of multiple indicators of stress in an archaeological sample from St. Peter's Church, Barton-on-Humber to investigate the impact of environmental and economic change on childhood development in two cemetery phases before and after AD1700, when generally better living conditions occurred. She found that some stress indicators, particularly vertebral neural canal (VNC) size, to show a positive impact of improved living conditions after AD1700 in childhood health between 5 and 14 years of age as

seen by increased neural canal diameters (Watts, 2013). However, other stress indicators such as linear enamel hypoplasia and cribra orbitalia (CO) (that together affect dental and skeletal development roughly before 6 years of age), did not show any difference between the cemetery phases, indicating that stress was still experienced at these earlier developmental ages (Watts, 2013). However Watts' (2013) analysis of stress indicators by adult-age mortality finds that those that experienced early life stress such as CO no longer experienced reduced adult lifespan after 1700AD, which suggests that with improved living conditions these early life stresses no longer impacted long term health and mortality (Watts, 2013).

Bioarchaeological studies that have used multiple indicators of stress to examine changes in development have also contributed more nuanced ways of interpreting phenotypic variation in stature, focusing not just on stunting but also on trajectories and tempo and the role of catch-up growth. When stress insults cause a slowing down of growth there is a potential for increased growth velocity to "catch up" to the original or maximum growth trajectory if there is a significant improvement in environmental and nutritional conditions (Tanner, 1981; Cameron, 2002). Alternatively, catch up growth can also occur even if the growth rate stays the same but individuals continue to grow for a longer period of time to achieve normal adult potential (Bogin, 1995; Stinson, 2000; Cameron, 2002). However, catch up growth is thought to be rare unless individuals are removed from the environment that was associated with the initial stress insult during early development (Stinson, 2000) and it is also dependent on the age of onset in stunting and age when improvement in conditions occurs (Martorell, 1989). Using transport documents of enslaved Africans, Steckel (1987) found growth stopped in males at 21 years of age and females at 19 years of age, with both sexes achieving normal stature exceeding the 25th centile as adults, despite falling below the first or second centile of modern height standards as children. Steckel (1987) credits this catch up growth, despite poor diet and disease load during childhood, due to the improved diet as young adolescents were forced into labor.

Catch up growth has been discussed more extensively in recent bioarchaeological studies. For example, a second study by Watts (2011) on VNC and adult stature in a medieval population from Fishergate House, York found small VNC size to correlate with early adult mortality in both sexes, as well as selective mortality with reduced stature in females. Watts (2011) suggests that both sexes who died in early adulthood experienced childhood age stress that affected their neural canal development, but that males were not significantly affected by physiological insults that occurred in later age as they still achieved normal stature. This study highlights the importance of catch-up growth that can occur with improvement in environmental conditions, and demonstrates it may differ between the sexes, perhaps related to different gender-based biocultural buffering mechanisms. Temple (2008) also discusses catch-up growth in his comparative study of stress indicators and phenotypic variation in prehistoric Japan. The author made a comparison of dental defects and stature between Jomon foragers from eastern and western Japan, the latter region showing more resource scarcity and environmental stressors in prehistory. While foragers from western Japan showed higher frequency of enamel hypoplasia, there was no difference in stature between the two regions, and Temple (2008) suggests catch up growth may have occurred after the childhood stress episodes. Importantly, this study considers the issue of severity in stress insults. Temple (2008) argues that early childhood stress that results in indicators such as enamel defects could represent simple episodic events that may not affect long-term phenotypic variation in stature that is a highly canalized trait. Stature was seen to decrease temporally, with stature decreasing between the Middle and Late and Final Jomon phases that is interpreted as reflecting chronic bouts of systemic stress such as infection and dietary stress (Temple, 2008). Klaus and Tam (2009) also examined indicators of systemic stress in the indigenous Mochica population of Morrope from the Northern coast of Peru, finding an increase in markers of stress, including porotic hyperotosis and reduced long bone growth, in children potentially stemming from increases in infectious disease rates after colonial contact. Klaus and Tam (2009), however, note a decrease in LEH that they interpret as reflecting high epidemic mortality, and also suggest catch-up growth as a factor because terminal adult stature was not significantly reduced.

In another recent comparative study of stress indicators and stature in Holocene foragers of the South African Cape, Pfeiffer and Harrington (2010) argue that Holocene foragers, while small in overall stature, do not show growth stunting and show a tempo of normal healthy growth. The skeletal individuals examined do show evidence for early developmental insults with the presence of both cribra orbitalia and growth arrest lines (Harris lines), but the authors suggest that these stress indicators paradoxically indicate that children were growing well and surviving in a challenging environment (Pfeiffer and Harrington, 2010). In this case, the authors point that the phenotypic signature of small adult body size was maintained throughout the Holocene by these ancestors of the KhoeSan groups by direct selection for lighter and smaller bodies, perhaps for energetic and environmental factors (Pfeiffer and Harrington, 2010, 2011). This study is highly relevant in pointing out the complex interpretation of evolutionary, developmental and environmental factors needed to evaluate phenotypic variation in stature in the past.

Many contemporary bioarchaeological studies that have used multiple stress indicators in a life course approach also address the concerns of what is termed "The Osteological Paradox," particularly the issues of selective mortality and heterogeneous frailty. The Osteological Paradox, first coined by in the seminal article by Wood et al. (1992) laid bare the biased nature of pathological indicators we examine in archaeological skeletal samples. Selective mortality refers to fact that the skeletal data we use come from assemblages that are biased representatives of the once-living population (Wood et al., 1992; DeWitte and Stojanowski, 2015). Hidden heterogeneity in frailty refers to fact that individuals are unequal in their susceptibility to stress and disease, and risk of death (Wood et al., 1992; DeWitte and Stojanowski, 2015). Selective mortality acts on differential frailty, so that individuals with the highest frailty at a particular age are the most likely to die and end up in the skeletal assemblage; as such they are not representative of all the individuals of that age in the entire living population (Wood et al., 1992; DeWitte and Stojanowski, 2015). A recent study by Vercelotti et al. (2014)

specifically highlights the role of the Osteological Paradox and caution required in interpreting early life stress and stature. Vercelotti et al. (2014) examined adult stature in two European medieval skeletal samples that experienced varying degrees of stress in comparison with stature growth in living populations from South America. In the archaeological populations examined, the authors found the medieval sample from Giecz, Poland with the greater frequencies of stress indicators (porotic hyperstosis and LEH) had taller stature as compared with the medieval sample from Italy (Trino Vercellese), contrary to what was expected (Vercellotti et al., 2014). Vercelloti et al. (2014) do not imply that stress had no impact on adult stature, but suggest that the stature of the surviving group might be skewed due to high selective pressures that eliminated short-statured individuals in the population that experienced systemic stress. At the same time the authors suggest this allowed catch-up growth for those that survived with improved adult diet (Vercellotti et al., 2014). As such, tall stature in the Giecz assemblage paradoxically indicates selective pressures rather than positive early life conditions (Vercellotti et al., 2014). A particularly interesting finding was the intertwined gender and status-related patterns of stress and stature within the Trino Verecellese sample. While high status in males was associated with lower LEH and taller stature, no such association was found in females, although low status females did show differences in LEH. The authors suggest this pattern could be attributed to differential biological buffering with influential factors such as diet, with low status adult males receiving poor diet (Vercellotti et al., 2014). This study is also unique in comparing patterns of growth with those in living populations that generally parallel the patterns of stunting with inequality in the archaeological populations.

The majority of studies of skeletal assemblages that have discussed selective mortality and/or frailty have primarily examined dental enamel defects. As mentioned earlier, a number of studies have found a relationship between early mortality and the presence of dental enamel defects (Goodman et al., 1988; Duray, 1996; Steckel, 2005; Boldsen, 2007; Miszkiewicz, 2015). However, in recent years researchers have considered not just the link between selective mortality and stress indicators but also been explicit in trying to link stress data to test the validity of the Barker and DOHaD Hypotheses (Armelagos et al., 2009; Ahlström, 2011; Weisensee, 2013; Amoroso et al., 2014; Temple, 2014). Armelagos et al. (2009) was one of the first to test the validity of the Barker Hypothesis using enamel defects in archaeological skeletal samples. In another sophisticated study in the Late/Final Jomon period Temple (2014) used LEH indicators and incremental microstructures of enamel to test two theories of early life stress, the predictive adaptive response (PAR) and the plasticity/constraint theory discussed earlier. Temple (2014) hypothesized that if enamel defects reflected a PAR response, then there would be no relationship between age-at-death and early life stress measured by LEH that would indicate the Jomon in this time period were able to resist future stress events. In contrast, he hypothesized if the plasticity/constraint model is a better fit, then individuals who experienced early life stress as indicated by LEH would be less effective at future investment in growth and maintenance due to the mitigation of early life stress (Temple, 2014). Temple (2014) found that individuals with dental defects that occurred early in life were at a greater risk of forming defects again at later stages and of dying at younger ages, in support of the plasticity/constraint model that suggests the plasticity in development observed is a life history trade-off. This study adds to the previous bioarchaeological studies that have shown a clear relationship between early life stress and early mortality. Interestingly, Temple (2014) found that growth disruptions that occurred early were the ones that affected future risk of stress disruption and risk of mortality, suggesting that these early stress indicators could have occurred at a critical developmental period that had greater long-term consequences. Amoroso et al. (2014) however, found conflicting support for the DOHaD hypothesis in their study of early mortality, LEH and socioeconomic-related quality of life in a modern skeletal sample (19th–20th century) from Lisbon. They found that while the presence and severity of LEH was related to early mortality, socioecomic circumstance accounted for most of the association with premature mortality (Amoroso et al., 2014). However, a study of another indicator of early life stress in the Lisbon collection, craniofacial fluctuating asymmetry, showed a positive association between those who died from degenerative diseases and higher asymmetry (Weisensee, 2013), perhaps pointing again to the complexity of interpreting early life stress. The study of stress indicators, their relationship to growth disruption, and their role in adult bone morphology, as well as overall morbidity and mortality, is clearly complicated. While these studies suggest caution in interpretation, the most recent work also demonstrates the contribution of bioarchaeological studies to explore the validity of life course theories.

## Life course approaches to diet in the past

In recent years there have also been studies of paleodiet and paleomobility that have taken a life course approach. While trace elemental and stable isotopic analyses have become widespread methods for reconstructing diet in past humans, recent studies have used isotope signatures from different parts within bones and teeth to reconstruct diet change over the life course. While these studies examine the variability of bone chemical signatures stored in the skeleton over the life course rather than strict bone morphology, they have great potential to give longitudinal dietary data that are clearly relevant to contextualizing observed bone growth and structural morphology in the past. More importantly, the goal of these studies is to gain a fuller understanding of individual life histories that can simultaneously be used to assess the role of biocultural and environmental influences on past communities.

Stable isotopes of carbon and nitrogen are the most widely used in isotopic analyses of paleodiet. In brief, stable carbon isotopes in plants vary according to the photosynthetic pathway they use. Animals that consume plant matter will incorporate a portion of the carbon from the plant food source into their tissues. Terrestrial plants utilize one of three photosynthetic pathways, and the carbon products that result from these systems have variable stable isotope ratios (Knudson et al., 2012). Most terrestrial plants (that include most cereals, legumes, vegetables, nuts, and fruits) use the $C_3$ pathway, or Calvin-Benson, while mainly tropical grasses (like maize, millet, and sugarcane) use the $C_4$, or

Hatch-Slack photosynthetic pathway (Katzenberg, 2000; Tykot, 2006; Schoeninger, 2011). The third pathway is the CAM (Crassulacean Acid Metabolism) pathway that results in $\delta^{13}C$ values intermediate between $C_3$ and $C_4$ pathways which includes cacti and succulents (Ambrose, 1993; Katzenberg, 2000; Schoeninger, 2011; Knudson et al., 2012). Carbon isotope values also differ significantly between organisms from marine versus terrestrial ecosystems as a consequence of dramatic differences in the carbon sources of these two reservoirs (Katzenberg, 2000; Schoeninger, 2011; Knudson et al., 2012). The carbon isotope values in the hydroxylapatite portion of bones and teeth reflect the carbon sources from all plants and animals in the whole diet, and carbon values from the organic collagen portion of bones and teeth tend to disproportionately represent carbon that is derived from protein sources in the diet (Ambrose and Norr, 1993; Schoeninger, 2011). As such, analysis of carbon isotope ratios from both the collagen and the hydroxyapatite component give more complete information about the full dietary composition (Ambrose, 1993; Schoeninger, 2011). Nitrogen values are derived from the collagen portions of bone and dentine in teeth and are linked to consumed protein sources. Stable nitrogen isotopes vary according to trophic level, and in general levels of $\delta^{15}N$ are higher in marine organisms vs. terrestrial organisms, and as such are useful for identifying marine food consumption, although the contribution of marine foods in an individual's diet would increase both the $\delta^{15}N$ and $\delta^{13}C$ values of bone collagen (Schoeninger and DeNiro, 1984; Schoeninger, 2011; Knudson et al., 2012). Nitrogen isotopes also are used in the reconstruction of breastfeeding and weaning practices in the past as children being breastfed have $\delta^{15}N$ values approximately three parts per million higher than their mothers that begin to decrease at the given rate of weaning, eventually leveling off to the value of the solid diet (Schoeninger and DeNiro, 1984; Fogel et al., 1989; Tsutaya and Yoneda, 2015). In addition there have been studies of sulfur, hydrogen, strontium, and oxygen isotopes in recent years (Schoeninger, 2011; Tsutaya and Yoneda, 2015), with stable oxygen isotopes and radiogenic strontium isotopes being used for studies of paleomobility (Knudson, 2009; Schoeninger, 2011; Knudson et al., 2012).

Hundreds of archaeological studies have utilized isotopic analyses to examine questions related to diet in the past, and more recent studies have used isotopic analyses to address larger anthropological questions concerning social status, gender, forensics, and diet throughout human evolution (for detailed reviews of isotopic analyses see Schoeninger and DeNiro, 1984; Schwarcz and Schoeninger, 1991; Ambrose, 1993; Katzenberg, 2000; Ambrose and Krigbaum, 2003; Lee-Thorp and Sponheimer, 2006; Schoeninger, 2011).Isotopic studies that have particularly used a life course approach have become increasingly common. Many have used isotope analysis to understand early diet and growth, particularly the study of weaning age. Differences in nitrogen isotopes in bone collagen and oxygen isotopes in tooth enamel between individuals of different ages have been used to determine age at weaning in the past (e.g. Katzenberg and Pfeiffer, 1995; Herring et al., 1998; Schurr, 1998; Wright and Schwarcz, 1998; Dupras et al., 2001; Pearson et al., 2010; Tsutaya and Yoneda, 2015). More recently, there has been an effort to extend this approach to examine not only dental tissue formed in

early growth and childhood, but also tooth and bone formed at differing periods or rates to build a life history perspective on past diets. Tooth enamel is made up of primarily hydroxyapatite, and forms in childhood and does not undergo subsequent remodeling. Primary dentine that makes up a significant amount of the tooth is high in collagen and is also formed mostly in childhood (Sealy et al., 1995; Sandberg et al., 2014). A small amount of secondary dentine is added near the pulp cavity later in life, and tertiary dentine can be added in the case of injury (Sealy et al., 1995). The adult secondary dentition begins to form at birth, with the molars starting mineralization at birth and complete at about 11 to 12 years of age, while the final third molars start to form at 7 years of age with roots completing growth in the early 1920s (Sealy et al., 1995; Hillson, 2005). As such, isotopic analyses in the dental enamel and dentine can provide information from childhood and early life. Bone however, models and remodels during life in response to physiological needs and biomechanical stress. Further, rates of bone turnover also vary across the skeleton depending on stress levels and/or composition of trabecular versus cortical tissues (Stout and Lueck, 1995; Robling and Stout, 2004). While bone tissue can reflect mineral and collagen material accumulated over a decade, cortical bone in long bone shafts can have slower remodeling as compared with skeletal elements with high turnover in cancellous bone that reflect bone tissue in recent years of life (Sealy et al., 1995; Stout and Lueck, 1995). As such, isotope analyses of different skeletal elements and teeth have the potential to record diet or residence patterns along different points of the life course and provide a type of "longitudinal" dietary data.

White (1991) made the first life history study of isotopic signatures from multiple tissues in Nubian mummies. Sealy et al. (1995) subsequently used five South African archaeological skeletons to examine stable isotope signatures from teeth as compared with bone, and by looking at skeletal elements with different rates of remodeling. The use of isotopic signatures in different teeth and comparisons of both enamel and dentine has allowed more detailed information on the process of breastfeeding practices in the past including the age of weaning, the introduction of solid foods, and duration of weaning. For example, Wright and Schwarz (1998) made foundational studies on the difference in carbon and oxygen isotopes in enamel between teeth within skeletons, as well as a comparative study of these signatures in tooth enamel with carbon and nitrogen signatures in dentine in a Prehispanic sample from Kaminlijuyu, Guatemala (Wright and Schwarcz, 1999). Dupras and Tocheri's (2007) study of weaning age in Roman period skeletons from Dakhleh Oasis, Egypt also used both enamel and dentine samples, examining differences in isotopes in utero, postbirth, pre and postweaning, as well as juvenile to adult ages. The use of both dental tissues allowed a longitudinal picture of isotopic signatures that suggests extended breastfeeding practices (Dupras and Tocheri, 2007). Approaches that examine isotopes in teeth in the life course as compared with conventional cross-sectional analyses are particularly noteworthy as they circumvent the issues associated with mortality bias and the danger of assessing weaning age from children that died during the process.

The few recent studies that have successfully extended the use of intratooth stable isotope analysis with

concurrent consideration of early stress indicators such as LEH relate most directly to the focus of this article. For example, Sandberg et al. (2014) conducted a study of dentine intratooth stable carbon and nitrogen isotope profiles in a small sample of adults from the medieval Nubian site of Kulubnarti in Sudan, and compared the profiles to the age-of-occurrence of LEH timing. The authors found that hypoplastic stress indicators occurred during weaning rather than before or after the weaning process. Interestingly, when their data was compared with previously published cross-sectional data that included bone isotopic signatures from juvenile and adult bone, the adult survivors appear to have been weaned earlier than nonsurvivors (Sandberg et al., 2014). The data suggests that individuals that relied on breastfeeding too long may have suffered from greater malnutrition stress in this population. More recent studies have also followed the early work by Sealy et al. (1995) and Cox and Sealy (1997) in examining isotopic profiles in multiple teeth along with samples of bone tissue. For example, Richards et al. (2002) utilized this life course approach to examine carbon and nitrogen isotopes of both rib bone and teeth to get a fuller picture of weaning age but also diet through childhood to adulthood. The authors found that both teeth and bone indicate that weaning occurred at about 2 years of age, and that weaning was relatively abrupt. Also, the diet in early/middle childhood as indicated by nitrogen isotopes appears different than in later years, possibly reflecting more plant based consumption in these childhood years. In a particularly sophisticated study of dental and bone isotopes, Reitsema and Vercelloti (2012) examined diet over the life course, and between status and sex subgroups in the medieval sample from Trino Vercellese, Italy. The results indicated a diet that was based on terrestrial resources and $C_4$ plants, but that low-status males differed from high-status males and females (Reitsema and Vercellotti, 2012). Interestingly, the authors note that the differences only develop in adulthood as childhood diets are all similar (Reitsema and Vercellotti, 2012). It is argued that there may have been a cultural buffer that gave females and children preferential access to better dietary foods, in agreement with the subsequent study by Vercelotti et al. (2014) on stress indicators in the same sample discussed earlier.

Studies of isotopic signatures in the past that use both dental and skeletal tissues are the most promising avenues for life course research, particularly in combination with other stress indicators or measures of bone morphology. Studies in smaller pilot samples and larger samples have shown methodological improvements in isotope analysis and continue to offer the possibility of tracking temporal relationships of breasfeeding and weaning along with later dietary patterns in single individuals, such as with tooth dentine serial sections (Fuller et al., 2003; Eerkens et al., 2011; Beaumont et al., 2013b; Eerkens and Bartelink, 2013; Burt and Garvie-Lok, 2013; Sandberg et al., 2014) laser ablation of enamel (Sandberg et al., 2012), or incremental sampling of hair keratin (Beaumont et al., 2013a). Jørkov et al. (2009) has also shown that the inner layers of the adult petrous part of the temporal bone can show diet in the fetal and early stages of life in a Medieval sample from Denmark, as this part of the skeleton is a small area that does not undergo any bone turnover after about 2 years of age. Bell et al. (2001) further developed the method of examining temporal isotopic signatures

from bone by examining different mineral densities in a single bony element using bone density fractionation along with stable isotope measurement, raising the exciting potential of gaining information on the last years down to even months before of an individual's death. Finally, strontium and stable oxygen isotopes can be used for evidence of paleomobility, and in conjunction with other isotopic analyses to build a life history approach to both diet and childhood residence (Chenery et al., 2010; Knudson et al., 2012). While there is still much to be learned about interpreting variation in isotopic signals within and between bones and teeth, and concern on particular issues such as isotopic trophic offsets in studies of weaning (Reynard and Tuross, 2015), the application of isotopic methods will continue to be key in life course studies in the past.

## Examining embodied life experience and the individual in bioarchaeology

The concept of the body as a product of both biological and social developmental context is central to developmental system theory approaches to plasticity and also in archaeological and epidemiological perspectives on embodied life experience. Ingold (1998) has argued that the body is a developmental system that is contextually dependent, and that more importantly humans grow and are active in their development through engagement with the social world. This social engagement can be both conscious (with agency) and/or unconscious (Krieger, 2001; White et al., 2009). Social anthropological discourse on embodiment has situated the body in its environmental context, and has strived to used concepts such as the "embedded body" and "local biologies" to highlight the importance of individual experience and external variables on creation of the body (Lock, 1993, 2015; Niewöhner, 2011), and most recently to even augment epigenetic interpretation (Lock, 2015). Important in these theoretical concepts of the body is the notion that bodies are dynamic and fluid, changing and created by what we do and experience over the lifetime (Ingold, 2013; Pálsson, 2013). Theoretical approaches of embodiment and the life course have been utilized in the analysis of mortuary data (Joyce, 2000; Meskell, 2000; Stoodley, 2000; Sofaer Derevenski, 2002; Crossland, 2010), but are not widely applied in the examination of skeletal data (Agarwal, 2012). In broader archaeology, scholars have emphasized the importance of a life course perspective in providing contextualization for the physical lifecycle (Gilchrist, 2000; Knudson and Stojanowski, 2008). There has also been a theoretical engagement in archaeology with ideas of individual agency and personhood (Dobres and Robb, 2000; Hamilakis et al., 2002; Fowler, 2004; Joyce, 2005), and more recently bioarchaeologists have taken this work more seriously to examine intersecting aspects of social identity from the skeletal body (Sofaer, 2006a; Knudson and Stojanowski, 2008; Agarwal, 2012). For example, Joanna Sofaer (2006b) and Rebecca Gowland (2006) emphasize the developmental process of gender over the life course, linking the study of gender with aging. Life course approaches such as these have also been used in recent years to consider aspect of aging and growing old (Robb, 2002; Gowland, 2009; Appleby, 2010), or childhood from archaeological skeletons (Perry, 2008; Redfern and Gowland, 2012; Barrett, 2014; Blom and Knudson, 2014; Ellis, 2014). Fundamentally, the emphasis in these

approaches is on the importance of social life in the creation of the biological skeleton. Sofaer (2006a) suggests viewing the skeleton as a form of material culture crafted through lived experience that blurs the division of the biological and social body. Studies that seek the integration of social theory into study of the skeleton, are still focused on bony indicators such as diet, stress and health, but do so with a humanistic focus on people the past with an eye to observe skeletons as a product of lived experience.

While the emphasis in bioarchaeology traditionally has been on variation in bone morphology or chemistry at the population level, there has been a contemporary resurgence of interest in the study of individual life histories through the examination of bony indicators of food consumption, activity patterns, disease experience and movement across biosocial landscapes. Importantly, these indicators are reconstructed alongside consideration of archaeological and archival contextual information as available (White et al., 2009) .Termed "osteobiographies," a word first coined by Frank Saul in 1961 (Saul et al., 1989), these bioarchaeological studies offer valuable insight to life course approaches in the past. There have been many osteobiographical studies of archaeological skeletons (for example Hawkey, 1998; Williamson and Pfeiffer, 2004; Mayes and Barber, 2008; Barrett and Blakey, 2011) and even a recent edited volume devoted to the approach (Stodder and Palkovich, 2012). While the focus is on individual skeletons, the goal of many of these studies is certainly to use data from the individual level to gain an understanding of skeletal variation at the population level, and to contribute to developing larger biosocial questions about the community (Robb, 2002).

A number of studies are noteworthy in specifically taking on a life course perspective to osteobiographies. For example Hawkey (1998) examines the progression of disability and pathology over the life course in her study of an individual from historic Gran Quivira Pueblo, New Mexico. Her approach is unique, focused not just on the disease, but the changes in joint and muscular movements and the correspondence to impairment over the life cycle. Similar osteobiographic accounts have been used in the study of activity and trauma patterns, and infectious disease on the skeleton over the lifecourse. For example, Heathcote et al. (2012) discuss violent trauma and evidence for arduous activity patterns that build from early life in a 16th to 17th century Chamorro man, set within the historical understanding of Chamorro "strong men" builders and stoneworkers. Similarly, Knüsel (2012) uses a life course approach in his impressive study of elbow injuries in a small skeletal sample of medieval men from England. Knüsel (2012) utilizes both skeletal and archival data, and detailed modern clinical data, to build a compelling understanding of how embodied behaviors that begin in young age and can lead to distinctive skeletal morphology that reflects how male identity changed and developed over the life course. Baker and Bolhofner (2014) use a contexulized approach to an early 15th century burial of a young woman from a basilica at Polis Chrysochous on Cyprus to show the biological and social effects of leprosy. Recent studies that combine bioarchaeological and biochemical methods to build osteobiographies are also particularly noteworthy. For example, Knudson et al. (2012) examined stable and radiogenic isotopes of enamel, bone and hair together with burial context to reconstruct diet and mobility over the lifetime in an isolated Andean individual buried along a pre-Columbian route in the Atacama Desert, Chile, an individual they suggest was likely regularly moving across a strategic trade/resource route. In another study by Torres-Rouff and Knudson (2007) of an individual buried in a larger cemetery in the San Pedro de Atacama oases, the authors use isotopic life history information in combination with skeletal indicators of lifestyle and activity, to demonstrate that despite foreign birth the man was fully acculturated into Atacameño society during his life. Douglas and Pietrusewky (2012) use both isotopic data and skeletal data from a Bronze age skeleton from Ban Chiang, Thailand to show the skeletal changes in an male immigrant who led a long life as a skilled craftsman. In another osteobiographic study by Wilson et al. (2007), the authors use both stable isotopes and aDNA evidence in four Andean child mummies to illuminate the preparatory stages and life of the children before their ritual killing.

While there is a sentiment that large sample size analyses in bioarchaeology allow more rigorous hypothesis testing, analysis at the individual level is critical if we are to understand fully the range of plasticity of the skeleton over the lifecourse. Analyses of individuals allow the detailed and nuanced exploration of morphology that does not always fall within the normal distribution of the larger community. What is key is the use of multiscalar analyses that utilize a comparative approach of examining both individual and population level bony signatures simultaneously, and make clear the larger anthropological questions sought in the osteobiographic study. Finally, critiques of the focus on the individual also note the imaginative exploration that is found in some analyses, as is particularly the case with the use of fictive narrative in osteobiography (Boutin, 2011, 2012). However, even narrative approaches are based within rigorous bioarchaeological analysis. Osteobiographies clearly provide a more experiential and humanistic approach to skeletal remains, but that does not mean they are less valuable than traditional biological approaches to understanding the plasticity and development of the skeleton over the lifecourse.

## BONE STRENGTH, MAINTENANCE, AND LOSS OVER THE LIFE COURSE

Studies of bone development and plasticity in archaeological samples have focused largely on growth, stress, and diet as discussed above. However, studies that have emphasized biomechanical and physiological influences on bone strength and loss in past populations are also central to life course approaches to bone morphology. The examination of long bone strength through the analysis of diaphyseal cross-sectional geometry is a widely used method in bioarchaeology to understand behavior, particularly changes in mobility in past populations. While most studies do not explicitly use a life course approach, the study of bone strength in archaeological samples examines the formation and maintenance of long bone shape and size over the lifetime, and emphasizes the cumulative influence of biomechanical strain that begins in early adolescence and adulthood. There have been many comparative temporal and geographic bioarchaeological studies of bone structural analyses, and studies have moved to a consideration of both biomechanical as well as nonmechanical influences on

morphological variation over the life course. There is also a greater concern toward understanding bone growth, remodeling and loss across the different bone envelopes, and how these change over the life course.

Long bone structural analyses of early hominids and modern humans are used to investigate a number of questions such as the role of climate on long bone structure and activity patterns. However, the majority of studies of diaphyseal bone structure have focused on questions related to past behaviors, such as changes in labor practices with shifts in economic or subsistence strategies. Studies have also noted differences in bone shape between the sexes that are suggested to be related to gendered differences in activity patterns in combination with sexual dimorphism in body shape (Ruff and Larsen, 2014). For example Ruff and Hayes (1983) showed that sex differences in lower limb structure for males and females from Pecos Pueblo related both to pelvis shape differences and to gendered activities where particular types of movements and associated biomechanical stress differed for Pecos men and women. More recently studies have considered how long bone structure changes over the life course, and intersects with other variables such as sex and status. For example, long bone analyses of samples from the Mayan site of Xcambó suggest that labor patterns changed for men through time and that women had similar strenuous workloads despite some women being "elite," suggesting a strong sexual division of labor that united women's work despite class differences (Maggiano et al., 2008). All of these studies underscore the importance of postnatal plasticity in bone shape and size. While caution has been noted over simplistic interpretations of long bone structural analyses (Lieberman et al., 2004; Pearson and Lieberman, 2004; Cowgill, 2010), comprehensive research in recent years has clarified the complexity of influences on long bone shape and size. In particular, research has shown that bone shape is a better indicator of mobility differences than bone size (Wescott, 2006; Ruff and Larsen, 2014), and that changes in shape and rigidity in the lower limb correspond to ontogenetic changes in loading related to learning to walk and gain mature gait patterns (Wescott, 2006; Cowgill et al., 2010). There have been only a few bioarchaeological investigations of variation and the trajectory in robusticity and strength in juveniles (Ruff et al., 1994; Wescott, 2006; Cowgill et al., 2010), however, a continued understanding of changes in bone shape and size over the life course is needed.

Recently there has been study of the temporal trends in bone structure and density. Ruff et al. (2015) examined temporal trends in upper and lower long bone strength in a large number of European skeletons dating from the Upper Paleolithic to the 20th century. The authors found a decline in mediolateral bending strength that is interpreted as reflecting the decline in mobility with the shift from foraging toward food production from foraging (Ruff et al., 2015). These findings are also consistent with the recent study of temporal changes in trabecular architecture in humans (Chirchir et al., 2015; Ryan and Shaw, 2015). Ryan and Shaw (2015) examined femoral trabecular architecture in four archaeological human populations representing foragers and sedentary agriculturalists, finding forager groups to have higher bone volume and thicker trabeculae that was interpreted as likely resulting from high physical activity at a young age. Chirchir et al. (2015) found low trabecular density

in limb epiphyses in recent modern humans as compared with extinct hominins, including pre-Holocene *Homo sapiens* that is also interpreted as relating to increased sedentism. While these studies indicate increased gracility of the modern human skeleton, it is unclear if this trend is primarily the result of decreased mechanical stimulus during development or also the result of direct selection for gracile and perhaps lighter skeletons as the selection pressure for more robust skeletons was lost with sedentary lifestyles (Martin, 2003; Chirchir et al., 2015). It should also be noted that not all human groups became strict sedentary food producers, some retained considerable mobility foraging or as pastoralists. Further, while these studies focus on changes in mid aged-adult bone, there was no direct consideration of sex-related changes or how these changes may have played out over the life course. A better understanding of the developmental interplay between various genetic and nonmechanical influences on skeletal robusticity is critical to examining adult bone morphology. Getting at changes on the different the bone envelopes, particularly endosteal versus periosteal surfaces, is certainly key to this understanding (Gosman et al., 2011; Ruff and Larsen, 2014). Studies indicate that the periosteal surface is more sensitive to mechanical influences during growth and before adolescence, whereas the endosteal surface in long bones is more sensitive to mechanical influences after mid-adolescence (Ruff et al., 1994; Gosman et al., 2011; Ruff and Larsen, 2014). It is important to understand bone remodeling and modeling processes and how material properties of bone change dynamically during growth and aging if we are to interpret the static bone morphology observed in the archaeological record (Currey, 2003; Agarwal and Beauchesne, 2011; Gosman et al., 2011). Two notable recent studies have attempted to consider both systemic and mechanical influences on bone structure and biology in archaeological samples. Temple et al. (2014) compared skeletal growth and stress between an Early Neolithic and Late Neolithic foragers from Cis-Bakal, Eastern Siberia, examining stature, cortical thickness, and medullary width. They found that the Early Neolithic sample, that previously showed skeletal evidence for systemic stress, exhibited stunting in femoral length and wasting in body mass, but no difference from the later Neolithic sample in cortical bone measures (Temple et al., 2014). The authors suggest that biomechanical strain may still have been sufficient in the early Neolithic despite systemic stress affecting growth (Temple et al., 2014). A study by Schug and Goldman (2014) also attempted to examine midshaft femoral bone morphology along with another indicator of bone turnover in the study of stress in a second millennium BC prehistoric sample of immature skeletons from India. The authors found that immature femora show alteration in cross-sectional bone shape that is consistent with the acquisition of locomotor behavior, as indicated by previous studies of ontogenetic changes noted earlier (Schug and Goldman, 2014). However, children during the first 10 years of life with low BMI, also show reduced compact bone mass consistent with a significant increase in cortical bone porosity (Schug and Goldman, 2014).

Such studies are important in demonstrating the complex interplay of systemic and mechanical influences on bone strength, morphology and quality over the life course. In the study of children from the prehistoric village of Inamgaon, India the extreme effects of wasting

and poor health on bone growth and metabolism could have had long term effects on adult bone morphology and health as suggested by life course theories in epidemiology discussed earlier (Schug and Goldman, 2014). Bioarchaeological studies that have examined bone remodeling and aging are also relevant to the study of plasticity in adult bone morphology. Studies of bone loss in bioarchaeology have focused primarily on the influences of nutrition and physical activity on bone maintenance (Agarwal, 2008), although there has been consideration of the influence of early growth and development on the mature skeleton in archaeological samples. The classic studies of bone loss in Sudanese Nubia were some of the first to consider and compare bone growth and maintenance in both juvenile and adult skeletons. Armelagos et al. (1972) suggested that femoral cortical bone loss in young-aged female Nubians was likely due to early growth disturbance and stress as young adults during pregnancy and lactation. A later study by Van Gerven et al. (1985) of cortical bone growth maintenance in juvenile Nubians from the Kulubnarti site found that while bone mineral content increased after birth, the process of modeling combined with likely periods of nutritional stress, caused a reduction in percent cortical area during early and late childhood. More recently studies have also focused on the structural variation of trabecular bone during growth. For example Kneissel et al. (1997) examined both ontogeny and aging patterns of trabecular bone in the vertebrae of a medieval Lower Nubian skeletal sample of juvenile and adults from Egypt. They found the trabecular bone volume to be greatest during adolescence when the juvenile rod-like trabeculae begin to change to plate-like structures (Kneissel et al., 1997). Kneissel et al. (1997) also noted that age-related loss of trabecular structure in adults occurred earlier than in modern populations. More recently, Gosman and Ketcham (2009) examined ontogenetic patterns in trabecular bone in the tibia in skeletons from the prehistoric Ohio Valley, noting changes in trabecular architecture from growth to skeletal maturity and with increasing ambulatory activities. A unique study by Rewekant (2001) correlated growth patterns and developmental stress with variation in skeletal morphology and bone loss. Rewekant (2001) examined the correlation of adult cortical bone loss with indicators of growth disturbance (specifically compression of the skull base and vertebral stenosis) in two Polish medieval populations with differing socioeconomic statuses. Greater metacarpal cortical bone loss was found in the population that also showed greater disturbance of bone growth during childhood (Rewekant, 2001). The population that appeared to have suffered greater environmental stress during growth also showed lower sexual dimorphism in measurements of metacarpal cortical bone and skull base height, suggestive of a relationship between the disturbance of growth and the achievement of peak bone mass, as well as later age- and sex-related patterns of bone loss (Rewekant, 2001)

Reproductive behavior is another factor that may influence the trajectory of bone maintenance and loss in older age. As suggested earlier, studies in the archaeological skeletons of Nubian females were thought to reflect pregnancy and lactation stress (Martin and Armelagos, 1979, 1985; Martin et al., 1984). Similarly, recent studies of low bone mineral density in young female medieval skeletons from Denmark (Poulsen et al., 2001) and Norway (Turner-Walker et al., 2001;

Mays et al., 2006) have emphasized that this is a result of insufficient nutrition together with pregnancy and lactation stress. However, applying a life course perspective, it is unclear what the influence of reproduction in young adulthood would have had on later adult bone mass and strength in these populations. For example, Vogel et al. (1990) study of a historical European skeletal sample found that female skeletons showed better trabecular connectivity when compared with modern populations, which was attributed to the benefits of high parity. While this seems contradictory to studies that suggest reproductive stress results in bone loss, it has been argued that the loss of bone during the reproductive years in women in the past was transitory, and that in many circumstances bone loss during reproduction would have little or no effect on long-term bone fragility in women who would have survived to old age (Agarwal and Stuart-Macadam, 2003; Agarwal et al., 2004; Agarwal, 2008; Agarwal and Grynpas, 2009).

These bioarchaeological studies emphasize the importance of early life experiences on the strength and fragility of the adult skeleton. While influences such as nutrition, physical activity, and reproduction are critical to understanding bone growth and maintenance, it is increasingly evident that what is key to understand are the trajectories these influences take over the life course in each individual and community (Fausto-Sterling, 2005; Agarwal and Beauchesne, 2011), and particularly how they affect bone metabolism along the different areas and bony envelopes of the skeleton (Robling and Stout, 2004; Peck and Stout, 2007; Gosman et al., 2011). An example of the complex influences on bone morphology, maintenance and fragility is seen in the well-studied British medieval skeletal sample, Wharram Percy. Mays et al. have found age- and sex-related loss of cortical bone mass and mineral density at Wharram Percy similar to modern populations (Mays, 1996; Mays et al., 1998). A later study by McEwan et al. (2005) then examined the correlation of radial bone mineral density (BMD) to indicators of growth disturbances (specifically cortical index (CI), Harris lines, and cribra orbitalia) typically attributed to poor nutrition, in juvenile skeletons from the sample. Bone mineral density was found to be correlated with overall growth but not strongly correlated with the cortical index (CI), the latter showing greater sensitivity to environmental stress (McEwan et al., 2005). The correlation with reduced cortical index and stress markers was also as seen in the prehistoric Nubian study by Van Gerven et al. (1985), discussed above. McEwan et al. (2005) suggest that perhaps bone mineral accrual is relatively stable despite environment (nutritional) stress as compared with cortical bone modeling, and a similar finding was also noted in the femur cortical index measurement (Mays, 1999). These studies suggest that long bone cortical thickness is strongly influenced by nutritional stress and that reduced bone quantity during modeling can be carried into adulthood acerbating later loss of bone density. However, the same population shows little or no evidence for typical fragility fracture (McEwan et al. 2005). Further, study of bone loss in the trabecular bone tissue of the vertebrae shows atypical patterns with bone loss in young age-females, and no significant difference in bone loss between the sexes in old age that has been suggested to reflect the role of reproductive factors in bone maintenance (Agarwal et al., 2004; Agarwal and Grynpas, 2009). Transitory bone loss would be expected in women of reproductive

age in the archaeological record as they were often likely pregnant or lactating at the time of death, while high parity and extended lactation may have conserved and strengthened bone post-menopause in the long (Agarwal et al., 2004; Agarwal and Grynpas, 2009). The patterns of bone maintenance and bone loss over the life course and in the differing bony tissues, suggests that bone loss Wharram Percy was not solely an outcome of aging and menopause.

While there have been numerous studies of bone aging and osteoporosis in bioarchaeology, the etiology of bone loss in the past remains unclear (Agarwal, 2008; Agarwal and Grynpas, 2009). In some regions archaeological samples show temporal and/or spatial patterns of bone loss, but many do not, and many populations in the past have different patterns of bone loss and fragility than those observed in modern Western populations (Agarwal, 2008). Like all bioarchaeological studies, those on bone loss in the past are problematic, and suffer from the use of often incomplete or biased skeletal samples, and the difficulties related to age determination in older individuals. However, the variable patterns of bone maintenance fragility in the past are in some ways not surprising given that groups in the past would have had very different biosocial histories from our own (Agarwal and Beauchesne, 2011). Studies of osteoporosis in the past deal with how much bone is laid down during growth, and how it is shaped and lost in later life. Bone maintenance and loss is the result of ontogenetic processes over the life course, with trajectories of bone maintenance laid out in early growth, refined during adulthood, and played out and modified within the everyday individual and generational choices of behavior and life experience (Agarwal and Beauchesne, 2011) (Fig.1). This perspective is grounded in the social concepts of mentioned earlier of the body being "embedded" in its biosocial environment (Niewöhner, 2011) and the creation of "local biologies" that are the result of a fluid and dynamic trajectory of development that is influenced by internal and external processes (Lock, 1993, 2015). If we observe only a snapshot of bone morphology (such as bone loss in only an older adult individual, only one area of the skeleton or using only one method) we end up with a skewed perspective on the complex and unique path that has created the observed bone morphology. The examination of bone quantity and quality is an ideal method to assess bone plasticity and development as it directly reflects the lived experience of the body crafted at the cellular level through bone remodeling. This approach views plasticity as more than adaptation to specific environmental contexts, and instead as a developmental process where plasticity is seen as constructing the body and skeleton over the lifecourse and potentially over generations of multiple life cycles, as suggested by the developmental systems theory (DST) models discussed earlier (Oyama, 2000b; Oyama et al., 2003; Fausto-Sterling, 2005; Agarwal and Beauchesne, 2011).

This theoretical approach has been supported by recent biomedical and epidemiological studies on bone mineral density and loss. Infant and adolescent growth spurts have been shown to be influential in defining later adult bone quality and quantity (Cooper et al., 2001, 2002, 2006; Miller, 2005; Javaid et al., 2006). While peak bone mass (the maximal amount of bony tissue accrued during growth) is generally thought to be inherited (Duncan et al., 2003), heritability of bone mass is complex, and is a fluid measure based on a relationship between
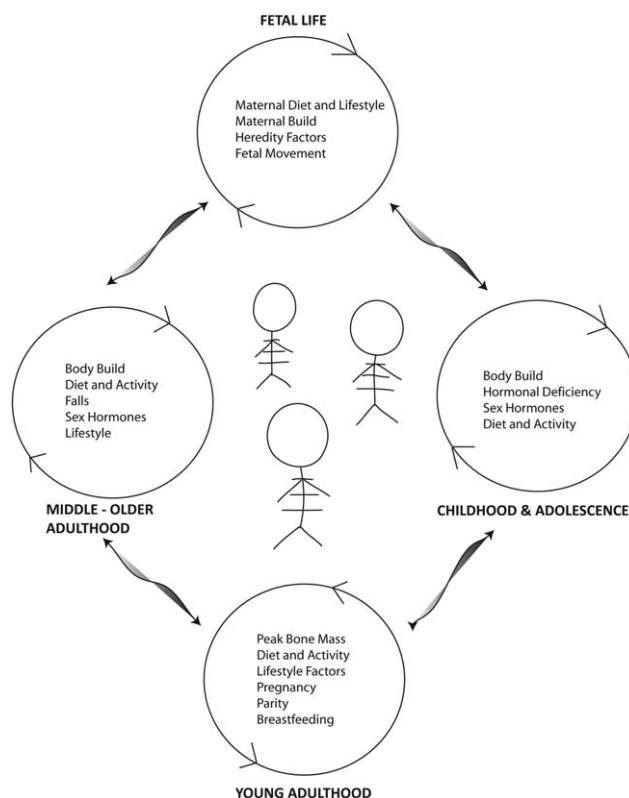


**Fig. 1.** A diagrammatic model of plasticity in development and maintenance of the skeleton over the life course. Bone maintenance and loss is the result of ontogenetic processes over the life course, with trajectories of bone maintenance laid out in early growth, refined during adulthood, and played out and modified within each individual and potentially generations. Circles represent major periods in the biological life cycle (fetal life, childhood and adolescence, young adulthood, and middle/older age) each containing examples of some of the major influences within each life stage in human skeletal development. Influences in each stage are cumulative and dependent on influences in earlier life stages (represented as arrows between circles). Cumulative influences shape overall skeletal morphology (depicted as the different skeletons in the center). (Figure adapted from Agarwal and Beauchesne 2011).

population and environment variance (Seeman, 1999). As age, height, gender, and body composition vary, so do heritability measures of bone mass or density (Seeman, 1999). Cooper et al. (2006) have suggested that fetal programming along with environmental cues early in life interact with the genome to create the boundaries of growth and development for a given individual. These developmental boundaries or trajectories may originate in the expectation of future environmental conditions, as outlined in theories such as the predictive adaptive response (PARS) (Gluckman and Hanson, 2004). Fetal programming by maternal under nutrition has been shown to be a risk factor for low birth weight (Cooper et al., 2002), and low birth weight is strongly correlated with lower levels of basal growth hormones placing individuals at risk for lower peak bone mass, reduced mineralization, and an elevated rate of bone loss later in life (Cooper et al., 2002; Dennison et al., 2005). Further, a number of studies have shown that impaired fetal and childhood growth place individuals at risk for fragility fractures later in life (Cooper et al., 2001, 2006;

Gale et al., 2001; Dennison et al., 2005). These studies on bone maintenance and later loss emphasize again the important role of environmental influences on phenotypic plasticity in early life, and how these early exposures can change the trajectory of development and aging of skeletal morphology throughout life.

## WORKING THROUGH THE LIMITATIONS AND GOING FORWARD WITH USING LIFE COURSE PARADIGMS IN BIOANTHROPOLOGY

Life course theory as a conceptual framework has been used in the biological and social sciences for some time. The epidemiological studies of the 1980s that first highlighted the consequences of stress in early fetal life (Barker's hypothesis), and later studies that have led to the more encompassing paradigm of the Developmental Origins of Health and Disease (DOHaD), have firmly established that events in early life play a central role together with genetic predispositions in influencing responses to the environment and later disease risk. Molecular epigenetics is providing potential explanatory mechanisms for how early life stress can be translated into disease risk in later life and even the potential for transgenerational inheritance. Finally, developmental systems theory (DST) approaches extend the concept to include ecological and social resources and epigenetic processes are seen as heritable across the life cycle. While it is unclear how nongenetic influences are inherited in relation to skeletal morphology, there is increasing discussion that nongenetic forces can shape postnatal and skeletal morphology significantly. While early human biologists and bioanthropologists pioneered many of the studies of early life stress and growth in human groups, the bones and teeth of past populations house unique information on developmental trajectories and histories. Contemporary bioarchaeologists have begun to extend and develop this work into sophisticated studies that attempt simultaneously to observe multiple indicators over the life course, directly test life course hypotheses, and challenged our ideas of variation in bone morphology.

A way in which researchers can continue to develop studies that are framed in a life course approach is to focus on multiple scales of analyses. This includes the examination of remodeling and modeling on differing bony envelopes as well as the use of multiple complementary methods in the same sample. Such research naturally leads to larger and more collaborative projects that often marry expertise in specialized methods such as histological, biomechanical, or isotopic analyses together with the examination of stress and pathology. The attention to multiple scales now also calls for a renewed interest in individual life histories and osteobiographies tied together with population level research. The consideration of influences on bone morphology over the life course is inherently difficult with the cross-sectional study of archaeological samples. It is particularly hard with the lack of precision in the determination of age at death in the oldest adults. Further, issues of heterogeneous frailty and selective mortality are particularly problematic when the goal is to assess stress in young age skeletons or morphology in old age. Studies that have explicitly considered the osteological paradox in their interpretation or have even circumvented these issues by conducting "longitudinal" studies within individual skeletons, such as isotopic studies of diet or bone remodeling over the life cycle, are particularly valuable. Further, the use of more rigorous biostatistical methods in bioarchaeological population studies that can better make probabilistic statements on prevalence are also needed. For example the use of odds ratios to estimate prevalence (Klaus and Tam, 2009; Klaus, 2014) or methods such as the person years construct to determine length of risk of exposure (Glencross and Sawchuk, 2003), offer the potential to be able to address problems related to age structures or proportional differences in sample sizes (Klaus, 2014).

Understanding how trajectories of bone growth and morphology can be altered and shaped over the life course is critical not only for bioarchaeologists, but also researchers studying bone morphology in living nonhuman primates and fossil primate skeletons. There has been extensive study of growth and development in fossil hominids, for example recently in post-cranial growth trajectories in *H. erectus* KNM-WT15000 (Graves et al., 2010) and numerous studies of dental development in living apes and fossil hominids (for example Beynon and Dean, 1988; Dean et al., 1993; Dean, 2010; Schwartz, 2012). However, the focus of these studies is to better understand phylogenetic relationships and variation in human patterns of growth. There has been some interest the possible influence of developmental stress in fossil primates. For example, White (1978) first identified enamel hypoplasia in a fossil hominid, and since then several subsequent studies of indicators of linear enamel hypoplasia have been examined in fossil hominids (Brunet et al., 2002; Guatelli-Steinberg, 2003, 2004; Guatelli-Steinberg et al., 2004) and Miocene primates (Lukacs, 2001). More recently a study by Martín-González et al. (2012) examined postcranial growth in Neanderthals, interpreting the slower growth in childhood as compared with modern humans as related to perhaps ontogenetic constraints and also metabolic stress, that they suggest could be responsible for the shorter stature observed in Neanderthals. There have also been many isotopic studies of diet in early hominids (Lee-Thorp and Sponheimer, 2006; Sponheimer et al., 2006, 2013; Ungar and Sponheimer, 2011; Lee-Thorp et al., 2012; Cerling et al., 2013) and also the study of weaning age from barium distribution in dental enamel in Neanderthals (Austin et al., 2013). Life course approaches with living nonhuman primate skeletal material has been equally limited, although there have been studies of stress markers such as LEH in nonhuman primates (Skinner, 1996; Swindler, 2002; Guatelli-Steinberg, 2003, 2004; Guatelli-Steinberg et al., 2004; Hannibal and Guatelli-Steinberg, 2005), and bone remodeling dynamics related to growth history that are geared towards understanding taxonomic and intra-skeletal patterns of cortical morphology (for example, McFarlin et al., 2008). While these studies in living and fossil primates are focused on aspects of biological *life history*, explicit and detailed *life course* approaches to analyses of fossil hominids and nonhuman primate material remain to be undertaken.

Our understanding of bone morphology and phenotypic variation in the past will continue to develop with increasing knowledge about bone metabolism and adaptation (Currey, 2003; Gosman et al., 2011), experimental studies of ontogenetic mechanisms in bone morphology (Hallgrimsson et al., 2002a; Roseman et al., 2010; Martínez-Abadías et al., 2012), and increasingly sophisticated molecular data in ancient bone (Briggs et al., 2010). While epigenetic influences on bone morphology

are not fully understood, they are clearly important as demonstrated by work on fetal programming in bone growth and loss, discussed earlier (Cooper et al., 2002, 2006; Dennison et al., 2005). Although we cannot empirically test how epigenetic mechanisms are at work in cross-sectional skeletal samples from the past (Klaus, 2014), we should not discount the role of early life experience on long term bone morphology. Recent studies that have tested the DOHaD hypothesis using archaeological skeletal samples (Clark et al., 1986; Armelagos et al., 2009; Watts, 2013; Temple, 2014) are contributing much to our understanding of developmental plasticity and disease epidemiology of the skeleton in both the past and the present. Gowland (2015) recently emphasized the implications of the DOHaD hypothesis in the interpretation of patterns of health in the bioarchaeological record, particularly as they relate to fetal and maternal health in the past. Similarly life course approaches have been important to understanding aspects of diet and bone chemistry (White and Schwarcz, 1994; Sealy et al., 1995; Dupras and Tocheri, 2007; Reitsema and Vercellotti, 2012; Gregoricka and Judd, 2015) and other influences on bone remodeling over the life cycle (Rewekant, 2001; Agarwal, 2012; Schug and Goldman, 2014; Temple et al., 2014). Similarly, studies that have utilized life course approaches in osteobiographical accounts (Boutin, 2011; Stodder and Palkovich, 2012) or in theoretical discussions of embodied life history (Fuentes, 2013 Ingold, 1998; Sofaer, 2006a) highlight the complex relationship between life experience and the physical body. These approaches are critical as we become increasingly aware that the chemical and morphological signatures of the skeletons we analyze are the result of life long and potentially even inter-generational creation.

Margaret Lock (2015) has called for an anthropological contribution to address the wider implications of molecular epigenetics. Bioarchaeologists have long been making this contribution—while studies of skeletal assemblages have only recently explicitly discussed epigenetics and utilized life course frameworks, bioarchaeology originated with the appreciation of humans as biosocial creations, not too unlike Pálsson's (2013) concept of humans embedded in "ensembles of biosocial relations." Data from isotopic signatures of diet, metabolic stress, and patterns of bone strength or bone loss in skeletal assemblages have been weaved together and have made significant contributions to understanding the biosocial landscapes for example of gender, inequality, violence, and aging in the past and present (Agarwal and Glencross, 2011; Martin et al., 2014). Bioarchaeologists have the opportunity to continue to make more deliberate contributions to understanding and interpreting postnatal plasticity. Phenotypic skeletal variation is the result of entangled biocultural trajectories of individual people that are embedded in the larger families and communities from which they are drawn. There is a resistance in biological anthropology towards approaches that are more humanistic or have roots in engagement with social theory. In order to reconstruct the rich and intricate life histories of human beings, however we need a holistic approach that uses our empirical testing of skeletal data as well as our imaginations.

## ACKNOWLEDGMENTS

## LITERATURE CITED

Agarwal S. 2008. Light and Broken Bones: Examining and Interpreting Bone Loss and Osteoporosis in Past Populations. In: Biological Anthropology of the Human Skeleton, Second. New York: Wiley-Liss. p (LC) 1–44.

Agarwal S., Beauchesne P. 2011. It is not carved in bone: development and plasticity of the aged skeleton. In: Agarwal S, Glencross B, editors. Social bioarchaeology. New York: Wiley-Blackwell. p 312–360

Agarwal SC. 2012. The past of sex, gender, and health: bioarchaeology of the aging skeleton. Am Anthropol 114:322–335.

Agarwal SC, Grynpas MD. 2009. Measuring and interpreting age-related loss of vertebral bone mineral density in a medieval population. Am J Phys Anthropol 139:244–252.

Agarwal SC, Stuart-Macadam P. 2003. An evolutionary and biocultural approach to understanding the effects of reproductive factors on the female skeleton. In: Agarwal SC, Stout SD, editors. Bone loss and osteoporosis: an anthropological perspective. New York: Kluwer Plenum Academic Press. p 105–116.

Agarwal S, Dumitriu M, Tomlinson G, Grynpas M. 2004. Medieval trabecular bone architecture: the influence of age, sex, and lifestyle. Am J Phys Anthropol 124(1):33–44.

Agarwal S., Glencross B. 2011. Building a social bioarchaeology. In: Agarwal S., Glencross B, editors. Social bioarchaeology. New York: Wiley-Blackwell.

Ahlström T. 2011. Life-history theory, past human populations and climatic perturbations. Int J Osteoarchaeol 21:407–419.

Allen HL, Estrada K, Lettre G, Berndt SI, Weedon MN, Rivadeneira F, Willer CJ, Jackson AU, Vedantam S, Raychaudhuri S, Ferreira T, Wood AR, Weyant RJ, Segrè AV, Speliotes EK, Wheeler E, Soranzo N, Park J-H, Yang J, Gudbjartsson D, Heard-Costa NL, Randall JC, Qi L, Smith AV, Mägi R, Pastinen T, Liang L, Heid IM, Luan J, Thorleifsson G, Winkler TW, Goddard ME, Lo KS, Palmer C, Workalemahu T, Aulchenko YS, Johansson Å, Zillikens MC, Feitosa MF, Esko T, Johnson T, Ketkar S, Kraft P, Mangino M, Prokopenko I, Absher D, Albrecht E, Ernst F, Glazer NL, Hayward C, Hottenga J-J, Jacobs KB, Knowles JW, Kutalik Z, Monda KL, Polasek O, Preuss M, Rayner NW, Robertson NR, Steinthorsdottir V, Tyrer JP, Voight BF, Wiklund F, Xu J, Zhao JH, Nyholt DR, Pellikka N, Perola M, Perry JRB, Surakka I, Tammesoo M-L, Altmaier EL, Amin N, Aspelund T, Bhangale T, Boucher G, Chasman DI, Chen C, Coin L, Cooper MN, Dixon AL, Gibson Q, Grundberg E, Hao K, Junttila MJ, Kaplan LM, Kettunen J, König IR, Kwan T, Lawrence RW, Levinson DF, Lorentzon M, McKnight B, Morris AP, Müller M, Ngwa JS, Purcell S, Rafelt S, et al. 2010. Hundreds of variants clustered in genomic loci and biological pathways affect human height. Nature 467:832–838.

Ambrose S. 1993. Isotopic analysis of paleodiets: methodological and interpretive considerations. In: Investigations of ancient human tissue: chemical analyses in anthropology. Gordon & Breach Science Publishers. Langhorne, PA. p 59–130.

Ambrose S, Krigbaum J. 2003. Bone chemistry and bioarchaeology. J Anthropol Archaeol 22:193–199.

Ambrose S, Norr L. 1993. Experimental evidence for the relationship of the carbon isotope ratios of whole diet and dietary protein to those of bone collagen and carbonate. In: Lambert, JB, Grupe, G, editors. Prehistoric human bone: archaeology at the molecular level. New York: Springer-Verlag. p 1–37.

Amoroso A, Garcia SJ, Cardoso HFV. 2014. Age at death and linear enamel hypoplasias: testing the effects of childhood stress and adult socioeconomic circumstances in premature mortality. Am J Hum Biol 26:461–468.

Appleby JEP. 2010. Why we need an archaeology of old age, and a suggested approach. Norwegian Archaeol Rev 43:145–168.

Armelagos G. 2003. Bioarchaeology as anthropology. Archaeol Pap Am Anthropol Assoc 13:27–40.

Armelagos GJ, Goodman AH, Harper KN, Blakey ML. 2009. Enamel hypoplasia and early mortality: bioarcheological support for the Barker hypothesis. Evol Anthropol Issues News Rev 18:261–271.

Armelagos GJ, Mielke JH, Owen KH, Van Gerven DP, Dewey JR, Mahler PE. 1972. Bone growth and development in prehistoric populations from Sudanese Nubia. J Hum E 1:89–119.

Austin C, Smith TM, Bradman A, Hinde K, Joannes-Boyau R, Bishop D, Hare DJ, Doble P, Eskenazi B, Arora M. 2013. Barium distributions in teeth reveal early-life dietary transitions in primates. Nature 498:216–219.

Baker BJ, Bolhofner KL. 2014. Biological and social implications of a medieval burial from Cyprus for understanding leprosy in the past. Int J Paleopathol 4:17–24.

Baker PT, Hanna JM, Baker TS, editors. 1986. The changing samoans: behavior and health in transition, 1st ed. New York: Oxford University Press.

Barker DJ, Osmond C. 1986. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. Lancet 1:1077–1081.

Barker DJ, Osmond C, Golding J, Kuh D, Wadsworth ME. 1989. Growth in utero, blood pressure in childhood and adult life, and mortality from cardiovascular disease. BMJ 298:564–567.

Barrett A. 2014. Childhood, colonialism, and nation-building: child labor in Virginia and New York. In: Thompson JL, Alfonso-Durruty MP, Crandall JJ, editors. Tracing childhood: bioarchaeological investigations of early lives in antiquity. Gainesville: University Press of Florida. p 183–197.

Barrett AR, Blakey ML. 2011. Life histories of enslaved Africans in colonial New York. In: Agarwal S, Glencross B, editors. Social bioarchaeology. New York: Wiley-Blackwell. p 212–251.

Baxter J. 1875. Statistics, medical, and anthropological, of over a million recruits. Washington, DC: Government Printing Office. Available from: https://archive.org/details/statisticsmedica02unit.

Beaumont J, Geber J, Powers N, Wilson A, Lee-Thorp J, Montgomery J. 2013a. Victims and survivors: stable isotopes used to identify migrants from the Great Irish Famine to 19th century London. Am J Phys Anthropol 150:87–98.

Beaumont J, Gledhill A, Lee-Thorp J, Montgomery J. 2013b. Childhood diet: a closer examination of the evidence from dental tissues using stable isotope analysis of incremental human dentine. Archaeometry 55:277–295.

Bell LS, Cox G, Sealy J. 2001. Determining isotopic life history trajectories using bone density fractionation and stable isotope measurements: a new approach. Am J Phys Anthropol 116:66–79.

Bengston V, Allen K. 1993. The life course perspective applied to families over time. In: Boss P, Doherty WJ, LaRossa R, Scham W, Steinmetz SK, editors. Sourcebook of families, theories and methods. New York: Plenum Press. p 469–498.

Benyshek DC. 2013. The "early life" origins of obesity-related health disorders: new discoveries regarding the intergenerational transmission of developmentally programmed traits in the global cardiometabolic health crisis. Am J Phys Anthropol 152:79–93.

Beynon AD, Dean MC. 1988. Distinct dental development patterns in early fossil hominids. Nature 335:509–514.

Blackwell DL, Hayward MD, Crimmins EM. 2001. Does childhood health affect chronic morbidity in later life? Soc Sci Med 1982 52:1269–1284.

Blom DE, Knudson KJ. 2014. Tracing Tiwanaku childhoods: a bioarchaeological study of age and social identities in Tiwanaku society. In: Thompson JL, Alfonso-Durruty MP, Crandall JJ, editors. Tracing childhood: bioarchaeological investigations of early lives in antiquity. Gainesville: University Press of Florida. p 228–245.

Boas F. 1912. Changes in the bodily form of descendants of immigrants. Am Anthropol 14:530–562.

Bogin B. 1995. Plasticity in the growth of Mayan refugee children living in the United States. In: Bogin B, Mascie-Taylor CG, editors. Human variability and plasticity. Cambridge studies in biological and evolutionary anthropology. Cambridge: Cambridge University Press. p 45–74.

Bogin B. 1999. Patterns of human growth, 2 ed. Cambridge: Cambridge University Press.

Bogin B, Rios L. 2003. Rapid morphological change in living humans: implications for modern human origins. Comp Biochem Physiol Part A 136:71–84.

Boldsen JL. 2007. Early childhood stress and adult age mortality–a study of dental enamel hypoplasia in the medieval Danish village of Tirup. Am J Phys Anthropol 132:59–66.

Boutin AT. 2011. Crafting a bioarchaeology of personhood: osteobiographical narratives from Alalakh. In: Baadsgaard A, Boutin AT, Buikstra JE, editors. Breathing New Life into the Evidence of Death: Contemporary Approaches to Bioarchaeology. Santa Fe, NM: School for Advanced Research Press. p 109–133.

Boutin AT. 2012. Written in stone, written in bone: the osteobiography of a bronze age craftsman from Alalakh. In: Stodder ALW, Palkovich AM, editors. The bioarchaeology of individual. Gainesville: University Press of Florida. p 193–214.

Bowditch HP. 1879. The growth of children: A supplementary investigation with suggestions in regard to methods of research. Boston, MA: Rand, Avery, printers to the Commonwealth.

Briggs AW, Stenzel U, Meyer M, Krause J, Kircher M, Pääbo S. 2010. Removal of deaminated cytosines and detection of in vivo methylation in ancient DNA. Nucleic Acids Res 38:e87–e87.

Brunet M, Fronty P, Sapanet M, de Bonis L, Viriot L. 2002. Enamel hypoplasia in a pliocene hominid from Chad. Connect Tissue Res 43:94–97.

Buikstra J, Cook D. 1980. Palaeopathology: an American account. Annu Rev Anthropol 9:433–470.

Buikstra JE, Beck LA, editors. 2009. Bioarchaeology: the contextual analysis of human remains. Walnut Creek, CA: Left Coast Press.

Burt NM, Garvie-Lok S. 2013. A new method of dentine microsampling of deciduous teeth for stable isotope ratio analysis. J Archaeol Sci 40:3854–3864.

Cameron N. 2002. Human growth curve, cnalization, and catch-up growth. In: Cameron N, editor. Human growth and development. Amsterdam: Academic Press. p 1–20.

Cerling TE, Manthi FK, Mbua EN, Leakey LN, Leakey MG, Leakey RE, Brown FH, Grine FE, Hart JA, Kaleme P, Roche H, Uno KT, Wood BA. 2013. Stable isotope-based diet reconstructions of Turkana Basin hominins. Proc Natl Acad Sci USA 110:10501–10506.

Chenery C, Müldner G, Evans J, Eckardt H, Lewis M. 2010. Strontium and stable isotope evidence for diet and mobility in Roman Gloucester, UK. J Archaeol Sci 37:150–163.

Chirchir H, Kivell TL, Ruff CB, Hublin J-J, Carlson KJ, Zipfel B, Richmond BG. 2015. Recent origin of low trabecular bone density in modern humans. Proc Natl Acad Sci USA 112:366–371.

Clark G, Hall N, Armelagos G, BORKAN G, Panjabi M, Wetzel F. 1986. Poor growth before early childhood: decreased health and life-span in the adult. Am J Phys Anthropol 70:145–160.

Clausen J. 1985. The life course: a sociological perspective, 1 ed. Englewood Cliffs, NJ: Pearson.

Cooper C, Eriksson JG, Forsén T, Osmond C, Tuomilehto J, Barker DJ. 2001. Maternal height, childhood growth and risk of hip fracture in later life: a longitudinal study. Osteoporos Int J Establ Result Coop Eur Found Osteoporos Natl Osteoporos Found USA 12:623–629.

Cooper C, Javaid MK, Taylor P, Walker-Bone K, Dennison E, Arden N. 2002. The fetal origins of osteoporotic fracture. Calcif Tissue Int 70:391–394.

Cooper C, Westlake S, Harvey N, Javaid K, Dennison E, Hanson M. 2006. Review: developmental origins of osteoporotic fracture. Osteoporos Int 17:337–347.

Cowgill LW. 2010. The ontogeny of Holocene and Late Pleistocene human postcranial strength. Am J Phys Anthropol 141:16–37.

Cowgill LW, Warrener A, Pontzer H, Ocobock C. 2010. Waddling and toddling: the biomechanical effects of an immature gait. Am J Phys Anthropol 143:52–61.

Cox G, Sealy J. 1997. Investigating identity and life histories: isotopic analysis and historical documentation of slave skeletons found on the Cape Town Foreshore, South Africa. Int J Hist Archaeol 1:207–224.

Crossland Z. 2010. Materiality and embodiment. In: Hicks D, Beaudry M, editors. The Oxford handbook of material culture studies (ms no. 19). Oxford: Oxford University Press. p 386–405.

Currey JD. 2003. The many adaptations of bone. J Biomech 36:1487–95.

Dean MC. 2010. Retrieving chronological age from dental remains of early fossil hominins to reconstruct human growth in the past. Philos Trans R Soc Lond B Biol Sci 365:3397–3410.

Dean MC, Beynon AD, Thackeray JF, Macho GA. 1993. Histological reconstruction of dental development and age at death of a juvenile Paranthropus robustus specimen, SK 63, from Swartkrans, South Africa. Am J Phys Anthropol 91:401–419.

Dennison EM, Syddall HE, Sayer AA, Gilbody HJ, Cooper C. 2005. Birth weight and weight at 1 year are independent determinants of bone mass in the seventh decade: the Hertfordshire cohort study. Pediatr Res 57:582–586.

DeWitte SN, Stojanowski CM. 2015. The Osteological Paradox 20 Years Later: Past Perspectives, Future Directions. J Archaeol Res 397–450.

DeWitte SN, Wood JW. 2008. Selectivity of Black Death mortality with respect to preexisiting health. Proc Natl Acad Sci USA 105:1436–1441.

Dias BG, Ressler KJ. 2014. Parental olfactory experience influences behavior and neural structure in subsequent generations. Nat Neurosci 17:89–96.

Dobres M-A, Robb J, editors. 2000. Agency in archaeology. London, NY: Routledge.

Douglas M, Pietrusewsky M. 2012. Vulcan: skilled village craftsman of Ban Chiang, Thailand. In: Stodder ALW, Palkovich AM, editors. The bioarchaeology of individuals. Gainesville: University Press of Florida. p 177–192.

Dupras TL, Schwarcz HP, Fairgrieve SI. 2001. Infant feeding and weaning practices in Roman Egypt. Am J Phys Anthropol 115:204–212.

Dupras TL, Tocheri MW. 2007. Reconstructing infant weaning histories at Roman period Kellis, Egypt using stable isotope analysis of dentition. Am J Phys Anthropol 134:63–74.

Duray SM. 1996. Dental indicators of stress and reduced age at death in prehistoric Native Americans. Am J Phys Anthropol 99:275–286.

Duncan EL, Cardon LR, Sinsheimer JS, Wass JAH, Brown MA. 2003. Site and gender specificity of inheritance of bone mineral density. J Bone Miner Res 18:1531–1538.

Eerkens JW, Bartelink EJ. 2013. Sex-biased weaning and early childhood diet among middle holocene hunter-gatherers in Central California. Am J Phys Anthropol 152:471–483.

Eerkens JW, Berget AG, Bartelink EJ. 2011. Estimating weaning and early childhood diet from serial micro-samples of dentin collagen. J Archaeol Sci 38:3101–3111.

Elder Jr G. 1995. The life course paradigm: Social change and individual development. In: Moen P, Elder Jr, Lüscher K, editors. Examining lives in context: perspectives on the ecology of human development. Washington, DC: American Psychological Association. p 101–139.

Elder Jr. G, Johnson MK, Crosnoe R. 2003. The emergence and development of life course theory. In: Mortimer JT, Shanahan MJ, editors. Handbook of the life course. Handbooks of sociology and social research. New York: Springer US. p 3–19.

Ellis M. 2014. A disciplined childhood in nineteenth century New York City: a social bioarchaeology of the subadults of the Spring Street Presbyterian Church publication. In: Thompson JL, Alfonso-Durruty MP, Crandall JJ, editors. Tracing childhood: bioarchaeological investigations of early lives in antiquity. Gainesville: University Press of Florida.

Ellison P., 2005. Evolutionary perspectives on the fetal origins hypothesis. Am J Hum Biol 17:113–118.

Fausto-Sterling A. 2005. The bare bones of sex: part 1-sex and gender. Signs J Women Cult Soc 30:1491–1527.

Fogel M, Tuross N, Owsley D. 1989. Nitrogen isotope tracers of human lactation in modern and archaeological populations. Washington, DC: Carnegie Institution of Washington.

Fowler C. 2004. The archaeology of personhood: an anthropological approach, 1 ed. New York: Routledge.

Fuentes A. 2013. Blurring the biological and social in human becomings. In: Biosocial Becomings: Integrating Social and Biological Anthropology. Cambridge: Cambridge University Press. p 42–58.

Fuller BT, Richards MP, Mays SA. 2003. Stable carbon and nitrogen isotope variations in tooth dentine serial sections from Wharram Percy. J Archaeol Sci 30:1673–1684.

Gale CR, Martyn CN, Kellingray S, Eastell R, Cooper C. 2001. Intrauterine programming of adult body composition. J Clin Endocrinol Metab 86:267–72.

Geber J. 2014. Skeletal manifestations of stress in child victims of the Great Irish Famine (1845–1852): prevalence of enamel hypoplasia, Harris lines, and growth retardation. Am J Phys Anthropol 155:149–161.

Gilchrist R. 2000. Archaeological biographies: realizing human lifecycles, -courses and -histories. World Archaeol 31:325–328.

Glencross B, Sawchuk L. 2003. The person years construct: ageing and prevalence of health related phenomena from skeletal samples. Int J Osteoarchaeol 6:369–376.

Gluckman PD, Hanson MA. 2004. The fetal matrix: evolution, development and disease. Cambridge: Cambridge University Press.

Goldstein MS. 1943. Demographic and bodily changes in descendants of Mexican immigrants,: With comparable data on parents and children in Mexico. TX: Institute of Latin-American studies, University of Texas.

Goodman A, Brooke Thomas R, Swedlund A, Armelagos G. 1988. Biocultural perspectives on stress in prehistoric, historical, and contemporary population research. Am J Phys Anthropol 31:169–202.

Gosman JH, Ketcham RA. 2009. Patterns in ontogeny of human trabecular bone from SunWatch Village in the Prehistoric Ohio Valley: general features of microarchitectural change. Am J Phys Anthr 138:318–332.

Gosman JH, Stout SD, Larsen CS. 2011. Skeletal biology over the life span: a view from the surfaces. Am J Phys Anthropol 146: 86–98.

Gowland R. 2006. Ageing the past: Examining age identity from funerary evidence. In: Gowland R, Knusel CJ, editors. Social archaeology of funerary remains. Oxford: Alden Press. p 143–153.

Gowland R. 2009. Age, ageism and osteological bias: the evidence from late Roman Britain. J Roman Archaeol Suppl Ser 65:153–16.

Gowland RL. 2015. Entangled lives: Implications of the developmental origins of health and disease hypothesis for bioarchaeology and the life course. Am J Phys Anthropol 158:530–540.

Gravlee CC. 2009. How race becomes biology: embodiment of social inequality. Am J Phys Anthropol 139:47–57.

Graves RR, Lupo AC, McCarthy RC, Wescott DJ, Cunningham DL. 2010. Just how strapping was KNM-WT 15000? J Hum Evol 59:542–554.

Gregoricka LA, Judd MA. 2015. Isotopic Evidence for Diet Among Historic Bedouin of Khirbat al-Mudayna, Jordan. Int J Osteoarchaeol. Article first published online: 14 JUL 2015 DOI: 10.1002/oa.2468

Guatelli-Steinberg D. 2003. Macroscopic and microscopic analyses of linear enamel hypoplasia in Plio-Pleistocene South

African hominins with respect to aspects of enamel development and morphology. Am J Phys Anthropol 120:309–322.

Guatelli-Steinberg D. 2004. Analysis and significance of linear enamel hypoplasia in Plio-Pleistocene hominins. Am J Phys Anthropol 123:199–215.

Guatelli-Steinberg D, Larsen CS, Hutchinson DL., 2004. Prevalence and the duration of linear enamel hypoplasia: a comparative study of Neandertals and Inuit foragers. J Hum Evol 47:65–84.

Hales CN, Barker DJP. 2001. The thrifty phenotype hypothesis type 2 diabetes. Br Med Bull 60:5–20.

Halfon N, Larson K, Lu M, Tullis E, Russ S. 2014. Lifecourse health development: past, present and future. Matern Child Health J 18:344–365.

Hallgrimsson B, Willmore K, Hall B. 2002a. Canalization, developmental stability, and morphological integration in primate limbs dedicated to the memory of Nancy Hong. Am J Phys Anthropol 119.

Hallgrimsson B, Willmore K, Hall BK. 2002b. Canalization, developmental stability, and morphological integration in primate limbs. Am J Phys Anthropol 119:131–158.

Hamilakis Y, Pluciennik M, Tarlow S. 2002. Thinking through the body: archaeologies of corporeality. New York: Springer Science & Business Media.

Hannibal DL, Guatelli-Steinberg D. 2005. Linear enamel hypoplasia in the great apes: analysis by genus and locality. Am J Phys Anthropol 127:13–25.

Hawkey DE. 1998. Disability, compassion and the skeletal record: using musculoskeletal stress markers (MSM) to construct an osteobiography from early New Mexico. Int J Osteoarchaeol 8:326–340.

Heathcote GM, Diego VP, Ishida HV, Sava J. 2012. An osteobiography of a remarkable protohistoric Chamorro man from Taga, Tinian. Micronesia 43:131–213.

Herring DA, Saunders SR, Katzenberg MA. 1998. Investigating the weaning process in past populations. Am J Phys Anthropol 105:425–439.

Hillson S. 2005. Teeth, 2 ed. New York: Cambridge University Press.

Holliday R., 2006. Epigenetics: a historical overview. Epigenet J DNA Methylation Soc 1:76–80.

Hoppa RD, FitzGerald CM, editors. 2005. Human growth in the past: studies from bones and teeth. Cambridge, NY: Cambridge University Press.

Humphrey L. 2000. Growth studies of past populations: an overview and an example. In: Cox, M, Mays, editors. Human osteology in archaeology and forensic science. Cambridge: Cambridge University Press. p 23–38.

Humphrey LT, King T. 2000. Childhood stress: a lifetime legacy. Anthropologie 38:33–49.

Ingold T. 1998. From complementarity to obviation: on dissolving the boundaries between social and biological anthropology, archaeology, and psychology. Cycles Conting Dev Syst Evol 123(1):255–279.

Ingold T. 2013. Prospect. In: Ingold T, Pálsson G, editors. Biosocial becomings: integrating social and biological anthropology. Cambridge: Cambridge University Press. p 1–21.

Jablonka E, Raz G. 2009. Transgenerational epigenetic inheritance: prevalence, mechanisms, and implications for the study of heredity and evolution. Q Rev Biol 84:131–176.

Javaid M, Lekamwasam S, Clark J, Dennison E, Syddall H, Loveridge N, Reeve J, Beck T, Cooper C. 2006. Infant growth influences proximal femoral geometry in adulthood. J Bone Miner Res 21:508–512.

Jørkov MLS, Heinemeier J, Lynnerup N. 2009. The petrous bone–a new sampling site for identifying early dietary patterns in stable isotopic studies. Am J Phys Anthropol 138:199–209.

Joyce RA. 2000. Girling the girl and boying the boy: the production of adulthood in ancient Mesoamerica. World Archaeol 31:473–483.

Joyce RA. 2005. Archaeology of the body. Annu Rev Anthropol 34:139–158.

Kamm K, Thelen E, Jensen JL. 1990. A dynamical systems approach to motor development. Phys Ther 70:763–775.

Kasl SV, Berkman L. 1983. Health Consequences of the Experience of Migration. Annu Rev Public Health 4:69–90.

Katzenberg MA. 2000. Stable isotope analysis: a tool for studying past diet, demography, and life history. In: Katzenberg MA, Saunders SR, editors. Biological anthropology of the human skeleton. New York: Wiley-Liss. p 305–328.

Katzenberg MA, Pfeiffer S. 1995. Nitrogen isotope evidence for weaning age in a nineteenth century Canadian skeletal sample. In: Grauer AL, editor. Bodies of evidence: reconstructing history through skeletal analysis. New York: Wiley-Liss. p 221–235.

Kemkes-Grottenthaler A. 2005. The short die young: the interrelationship between stature and longevity—evidence from skeletal remains. Am J Phys Anthropol 128:340–347.

Klaus HD. 2014. Frontiers in the bioarchaeology of stress and disease: cross-disciplinary perspectives from pathophysiology, human biology, and epidemiology. Am J Phys Anthropol 155:294–308.

Klaus HD, Tam ME. 2009. Contact in the Andes: bioarchaeology of systemic stress in colonial Mórrope, Peru. Am J Phys Anthropol 138:356–368.

Kneissel M, Roschger P, Steiner W, Schamall D, Kalchhauser G, Boyde A, Teschler-Nicola M. 1997. Cancellous bone structure in the growing and aging lumbar spine in a historic Nubian population. Calcif Tissue Int 61:95–100.

Knudson KJ. 2009. Oxygen isotope analysis in a land of environmental extremes: the complexities of isotopic work in the Andes. Int J Osteoarchaeol 19:171–191.

Knudson KJ, Pestle WJ, Torres-Rouff C, Pimentel G. 2012. Assessing the life history of an andean traveller through biogeochemistry: stable and radiogenic isotope analyses of archaeological human remains from Northern Chile. Int J Osteoarchaeol 22:435–451.

Knudson KJ, Stojanowski CM. 2008. New directions in bioarchaeology: recent contributions to the study of human social identities. J Archaeol Res 16:397–432.

Knüsel CJ. 2012. Men take up arms for war: sex and status distinctions of humeral medial epicondylar avulsion fractures in the archaeological record. In: Baadsgaard A, Boutin AT, Buikstra JE, editors. Breathing new life into the evidence of death. Santa Fe, NM: School for Advanced Research Press. p 221–250.

Krieger N. 2001. Theories for social epidemiology in the 21st century: an ecosocial perspective. Int J Epidemiol 30:668–677.

Krieger N. 2005. Embodiment: a conceptual glossary for epidemiology. J Epidemiol Commun Health 59:350–355.

Kuh D, Davey SG. 2004. The life course and adult chronic disease: an historical perspective with particular reference to coronary heart disease. In: Ben-Schlomo Y, Kuh D, editors. Life course approach to chronic disease epidemioloy, 2nd ed. Oxford: Oxford University Press. p 15–37.

Kuzawa CW. 2005. Fetal origins of developmental plasticity: are fetal cues reliable predictors of future nutritional environments? Am J Hum Biol Off J Hum Biol Counc 17:5–21.

Lai C-Q. 2006. How much of human height is genetic and how much is due to nutrition? Scientific American [Internet]. Available from: http://www.scientificamerican.com/article/how-much-of-human-height/.

Larsen CS. 2002. Bioarchaeology: the lives and lifestyles of past people. J Archaeol Res 10:119–166.

Larsen CS. 2015. Bioarchaeology: interpreting behavior from the human skeleton, 2nd ed. Cambridge, UK: Cambridge University Press.

Lasker G. 1976. Physical anthropology, 2nd ed. New York: Holt Rinehart and Winston.

Lasker GW. 1969. Human biological adaptability. The ecological approach in physical anthropology. Science 166:1480–1486.

Lasker GW, Evans FG. 1961. Age, environment and migration: further anthropometric findings on migrant and non-migrant Mexicans. Am J Phys Anthropol 19:203–211.

Leonard WR, Robertson ML. 1992. Nutritional requirements and human evolution: a bioenergetics model. Am J Hum Biol 4:179–195.

Lewis ME. 2009. The bioarchaeology of children: perspectives from biological and forensic anthropology, 1 ed. Cambridge: Cambridge University Press.

Lewontin RC. 2001. The triple helix: gene, organism, and environment. Cambridge, MA: Harvard University Press.

Lieberman DE, Polk JD, Demes B. 2004. Predicting long bone loading from cross-sectional geometry. Am J Phys Anthropol 123:156–171.

Little M, Kennedy K. 2010. Histories of American physical anthropology in the twentieth century. Lexington: Lanham.

Lock M. 2015. Comprehending the body in the era of the epigenome 1. Curr Anthropol 56:151–177.

Lock M. 1993. Encounters with aging: mythologies of menopause in Japan and North America. Berkeley: University of California Press.

Lovejoy CO, McCollum MA, Reno PL, Rosenman BA. 2003. Developmental biology and human evolution. Annu Rev Anthropol 32:85–109.

Low FM, Gluckman PD, Hanson MA. 2012. Developmental plasticity, epigenetics and human health. Evol Biol 39:650–665.

Lukacs JR. 2001. Enamel hypoplasia in the deciduous teeth of early Miocene catarrhines: evidence of perinatal physiological stress. J Hum Evol 40:319–329.

Maggiano IS, Schultz M, Kierdorf H, Sosa TS, Maggiano CM, Tiesler Blos V. 2008. Cross-sectional analysis of long bones, occupational activities and long-distance trade of the Classic Maya from Xcambó–archaeological and osteological evidence. Am J Phys Anthropol 136:470–477.

Martin DL, Armelagos GJ. 1979. Morphometrics of compact bone: an example from Sudanese Nubia. Am J Phys Anthr 51:571–577.

Martin DL, Armelagos GJ. 1985. Skeletal remodelling and mineralization as indicators of health: an example from prehistoric Sudanese Nubia. J Hum Evol 14:527–537.

Martin DL, Armelagos GJ, Goodman AH, Van Gerven DP. 1984. The effects of socioeconomic change in prehistoric Africa: Sudanese Nubia as a case study. In: Cohen MN, Armelagos GJ, editors. Paleopathology at the origins of agriculture. New York: Academic Press. p 193–214.

Martin DL, Harrod RP, Pérez VR. 2014. Bioarchaeology: an integrated approach to working with human remains, 2013 ed. New York: Springer.

Martínez-Abadías N, Mitteroecker P, Parsons TE, Esparza M, Sjøvold T, Rolian C, Richtsmeier JT, Hallgrímsson B. 2012. The developmental basis of quantitative craniofacial variation in humans and mice. Evol Biol 39:554–567.

Martín-González JA, Mateos A, Goikoetxea I, Leonard WR, Rodríguez J. 2012. Differences between Neandertal and modern human infant and child growth models. J Hum Evol 63:140–149.

Martin RB. 2003. Functional adaptation and fragility of the skeleton. In: Agarwal SC, Stout SD, editors. Bone loss and osteoporosis: an anthropological perspective. New York: Kluwer Plenum Academic Press. p 121–136.

Martorell R. 1989. Body size, adaptation and function. Hum Organ 48:15–20.

Mayer KU. 2009. New directions in life course research. Annu Rev Sociol 35:413–433.

Mayes AT, Barber SB. 2008. Osteobiography of a high-status burial from the lower Río Verde Valley of Oaxaca, Mexico. Int J Osteoarchaeol 18:573–588.

Mays SA. 1996. Age-dependent cortical bone loss in a medieval population. Int J Osteoarcheol 6:144–154.

Mays SA. 1999. Linear and oppositional long bone growth in earlier human populations. In: Hoppa RD, FitzGerald, editors. Human growth in the past: studies from bones and teeth. New York: Cambridge University Press. p 290–312.

Mays SA, Lees B, Stevenson JC. 1998. Age-dependent bone loss in the femur in a medieval population. Int J Osteoarchaeol 8:97–106.

Mays S, Brickley M, Ives R. 2008. Growth in an English population from the industrial revolution. Am J Phys Anthropol 136:85–92.

Mays S, Turner-Walker G, Syversen U. 2006. Osteoporosis in a population from medieval Norway. Am J Phys Anthropol 131:343–351.

Mcewan JM, Mays S, Blake GM. 2005. The relationship of bone mineral density and other growth parameters to stress indicators in a medieval juvenile population. Int J Osteoarchaeol 15:155–163.

McFarlin SC, Terranova CJ, Zihlman AL, Enlow DH, Bromage TG. 2008. Regional variability in secondary remodeling within long bone cortices of catarrhine primates: the influence of bone growth history. J Anat 213:308–324.

McHenry H. 1968. Transverse lines in long bones of prehistoric California Indians. Am J Phys Anthropol 29:1–17.

Meskell L. 2000. Cycles of life and death: narrative homology and archaeological realities. World Archaeol 31:423–441.

Miller M. 2005. Hypothesis: fetal movement influences fetal and infant bone strength. Med Hypotheses 65:880–886.

Miszkiewicz JJ. 2015. Linear enamel hypoplasia and age-at-death at medieval (11th–16th centuries) St. Gregory's priory and cemetery, Canterbury, UK. Int J Osteoarchaeol 25:79–87.

Niewöhner J. 2011. Epigenetics: embedded bodies and the molecularisation of biography and milieu. BioSocieties 6:279–298.

Oyama S. 2000a. Evolution's eye: a systems view of the biology-culture divide. Durham, NC: Duke University Press Books.

Oyama S. 2000b. The ontogeny of information: developmental systems and evolution, 2nd ed, Revised edition. Durham, NC: Duke University Press Books.

Oyama S, Gray RD, Griffiths PE, editors. 2003. Cycles of contingency: developmental systems and evolution, Reprint edition. Cambridge, MA: A Bradford Book.

Pálsson G. 2013. Ensembles of biosocial relations. In: Ingold T, Pálsson G, editors. Biosocial becomings: integrating social and biological anthropology. Cambridge: Cambridge University Press. p 42–58.

Pearson Ja, Hedges Rem, Molleson Ti, Özbek M. 2010. Exploring the relationship between weaning and infant mortality: an isotope case study from Aşıklı Höyük and Çayönü Tepesi. Am J Phys Anthropol 143:448–457.

Pearson OM, Lieberman DE. 2004. The aging of Wolff's "law": ontogeny and responses to mechanical loading in cortical bone. Am J Phys Anthropol 39:63–99.

Peck JJ, Stout SD. 2007. Intraskeletal variability in bone mass. Am J Phys Anthropol 132:89–97.

Perry MA. 2008. Redefining childhood through bioarchaeology: toward an archaeological and biological understanding of children in antiquity. Archeol Pap Am Anthropol Assoc 15:89–111.

Pfeiffer S, Harrington L. 2011. Bioarchaeological evidence for the basis of small adult stature in Southern Africa: growth, mortality, and small stature. Curr Anthropol 52:449–461.

Pfeiffer SK, Harrington L. 2010. Child growth among southern African foragers. In: Moffat T, Prowse T, editors. Human diet and nutrition in biocultural perspective. Oxford, NY: Berghahn Press. p 35–56.

Poulsen LW, Qvesel D, Brixen K, Vesterby A, Boldsen JL. 2001. Low bone mineral density in the femoral neck of medieval women: a result of multiparity? Bone 28:454–458.

Redfern R, Gowland R. 2012. A bioarchaeological perspective on the pre-adult stages of the life course: implications for the care and health of children in the Roman Empire. In: Harlow M, Loven, Lena L, editors. Families in the Roman and late antique world (family in antiquity). London: Continum International Publishing Group. p 110–139.

Reitsema LJ, Vercellotti G. 2012. Stable isotope evidence for sex- and status-based variations in diet and life history at medieval Trino Vercellese, Italy. Am J Phys Anthropol 148:589–600.

Rewekant A. 2001. Do environmental distrubances of an individual's growth and development influence the later bone involution processes? A study of two Mediaeval populations. Int J Osteoarchaeol 11:433–443.

Reynard LM, Tuross N. 2015. The known, the unknown and the unknowable: weaning times from archaeological bones using nitrogen isotope ratios. J Archaeol Sci 53:618–625.

Richards MP, Mays S, Fuller BT. 2002. Stable carbon and nitrogen isotope values of bone and teeth reflect weaning age at the Medieval Wharram Percy site, Yorkshire, UK. Am J Phys Anthropol 119:205–10.

Robb J. 2002. Time and biography. In: Hamilakis Y, Pluciennik M, Tarlow S, editors. Thinking through the body. New York: Springer US. p 153–171.

Robert JS, Hall BK, Olson WM. 2001. Bridging the gap between developmental systems theory and evolutionary developmental biology. BioEssays 23:954–962.

Roberts D. 1995. The pervasiveness of plasticity. In: Mascie-Taylor CGN, Bogin B, editors. Human variability and plasticity. Cambridge: Cambridge University Press. p 1–17.

Robling AG, Stout SD. 2004. Histomorphology, geometry, and mechanical loading in past populations. In: Agarwal SC, Stout SD, editors. Bone loss and osteolorosis: an anthropological perspective. New York: Kluwer Plenum Academic Press. p 184–201.

Roseman CC, Willmore KE, Rogers J, Hildebolt C, Sadler BE, Richtsmeier JT, Cheverud JM. 2010. Genetic and environmental contributions to variation in baboon cranial morphology. Am J Phys Anthropol 143:1–12.

Ruff CB, Hayes WC. 1983. Cross-sectional geometry of Pecos Pueblo femora and tibiae–a biomechanical investigation: II. Sex, age, side differences. Am J Phys Anthropol 60:383–400.

Ruff CB, Holt B, Niskanen M, Sladek V, Berner M, Garofalo E, Garvin HM, Hora M, Junno J-A, Schuplerova E, Vilkama R, Whittey E. 2015. Gradual decline in mobility with the adoption of food production in Europe. Proc Natl Acad Sci USA 112:7147–7152.

Ruff CB, Larsen CS. 2014. Long bone structural analyses and the reconstruction of past mobility: a historical review. In: Carlson KJ, Marchi D, editors. Reconstructing mobility: environmental, behavioral, and morphological determinants. New York: Springer. p 13–29.

Ruff CB, Walker A, Trinkaus E. 1994. Postcranial robusticity in Homo. III: Ontogeny. Am J Phys Anthropol 93:35–54.

Ryan T, Krovitz G. 2006. Trabecular bone ontogeny in the human proximal femur. J Hum Evol 51:591–602.

Ryan TM, Shaw CN. 2015. Gracility of the modern Homo sapiens skeleton is the result of decreased biomechanical loading. Proc Natl Acad Sci USA 112:372–377.

Sandberg PA, Sponheimer M, Lee-Thorp J, Van Gerven D. 2014. Intratooth stable isotope analysis of dentine: a step toward addressing selective mortality in the reconstruction of life history in the archaeological record. Am J Phys Anthropol 155:281–293.

Sandberg P, Passey P, Lee-Thorp J, Sponheimer M, Ditchfield P, Gerven DV. 2012. Intratooth stable isotope analysis of human dental tissues: laser ablation of enamel and serial sections of dentine collagen in permanent first molars and canines. Am J Phys Anthropol 147:257.

Saul F, Saul J, İşcan M, Kennedy K. 1989. Osteobiography: a maya example. In: Reconstruction of life from the skeleton. New York: Wiley-Liss. p 287–302.

Saunders S, Hoppa R. 1993. Growth deficit in survivors and non-survivors: biological mortality bias in subadult skeletal samples. Yearb Phys Anthropol 36:127–151.

Saunders SR. 2000. Subadult skeletons and growth-related studies. In: Katzenberg MA, Saunders SR, editors. Biological anthropology of the human skeleton. New York: Wiley-Liss. p 135–162.

Schell LM. 1995. Human biological adaptability with special emphasis on plasticity: history, development and problems for future research. In: Mascie-Taylor CGN, Bogin B, editors. Human variability and plasticity. Cambridge: Cambridge University Press. p 213–237.

Ben-Schlomo Y, Kuh D. 2002. A life course approach to chronic disease epidemiology: conceptual models, empirical challenges, and interdisciplinary perspectives. Int J Epidemiol 31:1–9.

Schoeninger M. 2011. Diet reconstruction and ecology using stable isotope ratios. In: Larsen C, editor. A companion to biological anthropology. Chichester: Wiley-Blackwell. p 445–464.

Schoeninger M, DeNiro M. 1984. Nitrogen and carbon isotopic composition of bone collagen from marine and terrestrial animals. Geochim Cosmochim Acta 48:625–639.

Schug GR, Goldman HM. 2014. Birth is but our death begun: a bioarchaeological assessment of skeletal emaciation in immature human skeletons in the context of environmental, social, and subsistence transition. Am J Phys Anthropol 155:243–259.

Schurr M. 1998. Using stable nitrogen isotopes to study weaning behavior in past populations. World Archaeol 30:327–342.

Schwartz GT. 2012. Growth, development, and life history throughout the evolution of Homo. Curr Anthropol 53:S395–S408.

Schwarcz HP, Schoeninger MJ. 1991. Stable isotope analyses in human nutritional ecology. Am J Phys Anthropol 34:283–321.

Scott AB, Hoppa RD. 2015. A re-evaluation of the impact of radiographic orientation on the identification and interpretation of Harris lines. Am J Phys Anthropol 156:141–147.

Sealy J, Armstrong R, Schrire C. 1995. Beyond lifetime averages: tracing life histories through isotopic analysis of different calcified tissues from archaeological human skeletons. Antiquity 69:290–300.

Seeman E. 1999. Genetic determinants of the population variance in bone mineral density. In: Rosen CJ, Glowacki J, Bilezikian JP, editors. The aging skeleton. San Diego: Academic Press. p 77–94.

Shapiro HL. 1939. Migration and environment. Oxford: Oxford University Press.

Skinner M. 1996. Developmental stress in immature hominines from Late Pleistocene Eurasia: evidence from enamel hypoplasia. J Archaeol Sci 23:833–852.

Sofaer Derevenski JR. 2002. Engendering context: context as gendered practice in the Early Bronze age of The Upper Thames Valley, UK. Eur J Archaeol 5:191–211.

Sofaer J. 2006a. The body as material culture: a theoretical osteoarchaeology. Cambridge: University of Cambridge Press.

Sofaer J. 2006b. Gender, bioarchaeology, and human ontogeny. In: Social archaeology of funerary remains. Studies in funerary archaeology 1. Oxford: Oxbow Books. p 155–167.

Sponheimer M, Alemseged Z, Cerling TE, Grine FE, Kimbel WH, Leakey MG, Lee-Thorp JA, Manthi FK, Reed KE, Wood BA, Wynn JG. 2013. Isotopic evidence of early hominin diets. Proc Natl Acad Sci USA 110:10513–10518.

Sponheimer M, Passey BH, de Ruiter DJ, Guatelli-Steinberg D, Cerling TE, Lee-Thorp JA. 2006. Isotopic evidence for dietary variability in the early hominin *Paranthropus robustus*. Science 314:980–982.

Steckel RH., 1987. Growth depression and recovery: the remarkable case of American slaves. Ann Hum Biol 14:111–132.

Steckel RH. 2005. Young adult mortality following severe physiological stress in childhood: skeletal evidence. Econ Hum Biol 3:314–328.

Stinson S. 2000. Growth variation: biological and cultural factors. In: Stinson S, Bogin B, Huss-Ashmore R, O'Rourke D, editors. Human biology: and evolutionary and biocultural perspective. New York: Wiley-Liss. p 425–264.

Stodder ALW, Palkovich AM, editors. 2012. The bioarchaeology of Individuals. FL: University Press of Florida.

Stoodley N. 2000. From the cradle to the grave: age organization and the early Anglo-Saxon burial rite. World Archaeol 31:456–472.

Stout SD, Lueck R. 1995. Bone remodeling rates and skeletal maturation in three archaeoloqical skeletal populations. Am J Phys Anthropol 98:161–171.

Swindler DR. 2002. Primate dentition: an introduction to the teeth of non-human primates. Cambridge: Cambridge University Press.

Tanner JM. 1981. Catch-up growth in man. Br Med Bull 37:233–238.

Temple DH. 2008. What can variation in stature reveal about environmental differences between prehistoric Jomon foragers? Understanding the impact of systemic stress on developmental stability. Am J Hum Biol 20:431–439.

Temple DH. 2010. Patterns of systemic stress during the agricultural transition in prehistoric Japan. Am J Phys Anthropol 142:112–124.

Temple DH. 2014. Plasticity and constraint in response to early-life stressors among late/final jomon period foragers from Japan: evidence for life history trade-offs from incremental microstructures of enamel. Am J Phys Anthropol 155:537–545.

Temple DH, Bazaliiskii VI, Goriunova OI, Weber AW. 2014. Skeletal growth in early and late Neolithic foragers from the Cis-Baikal region of Eastern Siberia. Am J Phys Anthropol 153:377–386.

Temple DH, Goodman AH. 2014. Bioarcheology has a "health" problem: Conceptualizing "stress" and "health" in bioarcheological research. Am J Phys Anthropol 155:186–191.

Lee-Thorp J, Likius A, Mackaye HT, Vignaud P, Sponheimer M, Brunet M. 2012. Isotopic evidence for an early shift to C4 resources by Pliocene hominins in Chad. Proc Natl Acad Sci USA 109:20369–20372.

Lee-Thorp J, Sponheimer M. 2006. Contributions of biogeochemistry to understanding hominin dietary ecology. Am J Phys Anthropol 131:131–148.

Torres-Rouff C, Knudson KJ. 2007. Examining the life history of an individual from solcor 3, San Pedro de Atacama: combining bioarchaeology and archaeological chemistry. Chungara, Revista De Antropología Chilena 39:235–256.

Tsutaya T, Yoneda M. 2015. Reconstruction of breastfeeding and weaning practices using stable isotope and trace element analyses: a review. Am J Phys Anthropol 156:2–21.

Turner BL, Armelagos GJ. 2012. Diet, residential origin, and pathology at Machu Picchu, Peru. Am J Phys Anthropol 149:71–83.

Turner-Walker G, Syverson U, Mays S. 2001. The archaeology of osteoporosis. J Eur Archaeol 4:263–268.

Tykot R. 2006. Isotope analyses and the histories of maize. In: History of maize. Burlington, MA: Academic Press.

Ungar PS, Sponheimer M. 2011. The diets of early hominins. Science 334:190–193.

Van Gerven DP, Hummert JR, Burr DB. 1985. Cortical bone maintenance and geometry of the tibia in prehistoric children from Nibia's Batn el Hajar. Am J Phys Anthropol 66:272–280.

Vercellotti G, Piperata BA, Agnew AM, Wilson WM, Dufour DL, Reina JC, Boano R, Justus HM, Larsen CS, Stout SD, Sciulli PW. 2014. Exploring the multidimensionality of stature variation in the past through comparisons of archaeological and living populations. Am J Phys Anthropol 155:229–242.

Vogel M, Hahn M, Caselitz P, Woggan J, Pompesius-Kempa M, Delling G. 1990. Comparison of trabecular bone structure in man today and an ancient population in Western Germany. In: Takahashi HE, editor. Bone morphometry. Japan: Nishimura Co. p 220–223.

Watts R. 2011. Nonspecific indicators of stress and their association with age at death in Medieval York: Using stature and vertebral neural canal size to examine the effects of stress occurring during different periods of development. Int J Osteoarchaeol 21:568–576.

Watts R. 2013. Childhood development and adult longevity in an archaeological population from Barton-upon-Humber, Lincolnshire, England. Int J Paleopathol 3:95–104.

Weisensee KE. 2013. Assessing the relationship between fluctuating asymmetry and cause of death in skeletal remains: a test of the developmental origins of health and disease hypothesis. Am J Hum Biol Off J Hum Biol Counc 25:411–417.

Wescott DJ. 2006. Effect of mobility on femur midshaft external shape and robusticity. Am J Phys Anthropol 130:201–213.

Wheeler SM. 2012. Nutritional and disease stress of juveniles from the Dakhleh Oasis, Egypt. Int J Osteoarchaeol 22:219–234.

White C. 1991. Isotopic Analysis of Multiple Human Tissues from Three Ancient Nubian Populations. Ph.D. Dissertation. University of Toronto.

White CD, Schwarcz HP. 1994. Temporal trends in stable isotopes for Nubian mummy tissues. Am J Phys Anthropol 93:165–187.

White C, Longstaffe FJ, Pendergast DM, Maxwell J. 2009. Cultural embodiment and the enigmatic identity of the lovers from Lamanai. In: Knudson KJ, Stojanowski CM, editors. Bioarchaeology and identity in the Americas. Tallahassee: University Press of Florida. p 155–176.

White TD. 1978. Early hominid enamel hypoplasia. Am J Phys Anthropol 49:79–83.

Williamson RF, Pfeiffer S, editors. 2004. Bones of the ancestors: the archaeology and osteobiography of the Moatfield Ossuary. Gatineau, Quebec: Canadian Museum of History.

Wilson AS, Taylor T, Ceruti MC, Chavez JA, Reinhard J, Grimes V, Meier-Augenstein W, Cartmell L, Stern B, Richards MP, Worobey M, Barnes I, Gilbert MTP. 2007. Stable isotope and DNA evidence for ritual sequences in Inca child sacrifice. Proc Natl Acad Sci USA 104:16456–16461.

Wood JW, Milner GR, Harpending HC, Weiss KM, Cohen MN, Eisenberg LE, Hutchinson DL, Jankauskas R, Cesnys G, Česnys G, Katzenberg MA, Lukacs JR, McGrath JW, Roth EA, Ubelaker DH, Wilkinson RG. 1992. The osteological paradox: problems of inferring prehistoric health from skeletal samples [and comments and reply]. Curr Anthropol 33:343–370.

Worthman C. 1995. Hormones, sex, and gender. Annu Rev Anthropol 24:593–617.

Worthman C, Kuzara J. 2005. Wiley-Liss plenary symposium life history and the early origins of health differentials. Am J Hum Biol 17:95–112.

Wright LE, Schwarcz HP. 1998. Stable carbon and oxygen isotopes in human tooth enamel: Identifying breastfeeding and weaning in prehistory. Am J Phys Anthropol 106:1–18.

Wright LE, Schwarcz HP. 1999. Correspondence between stable carbon, oxygen and nitrogen isotopes in human tooth enamel and dentine: infant diets at Kaminaljuyú. J Archaeol Sci 26:1159–1170.